

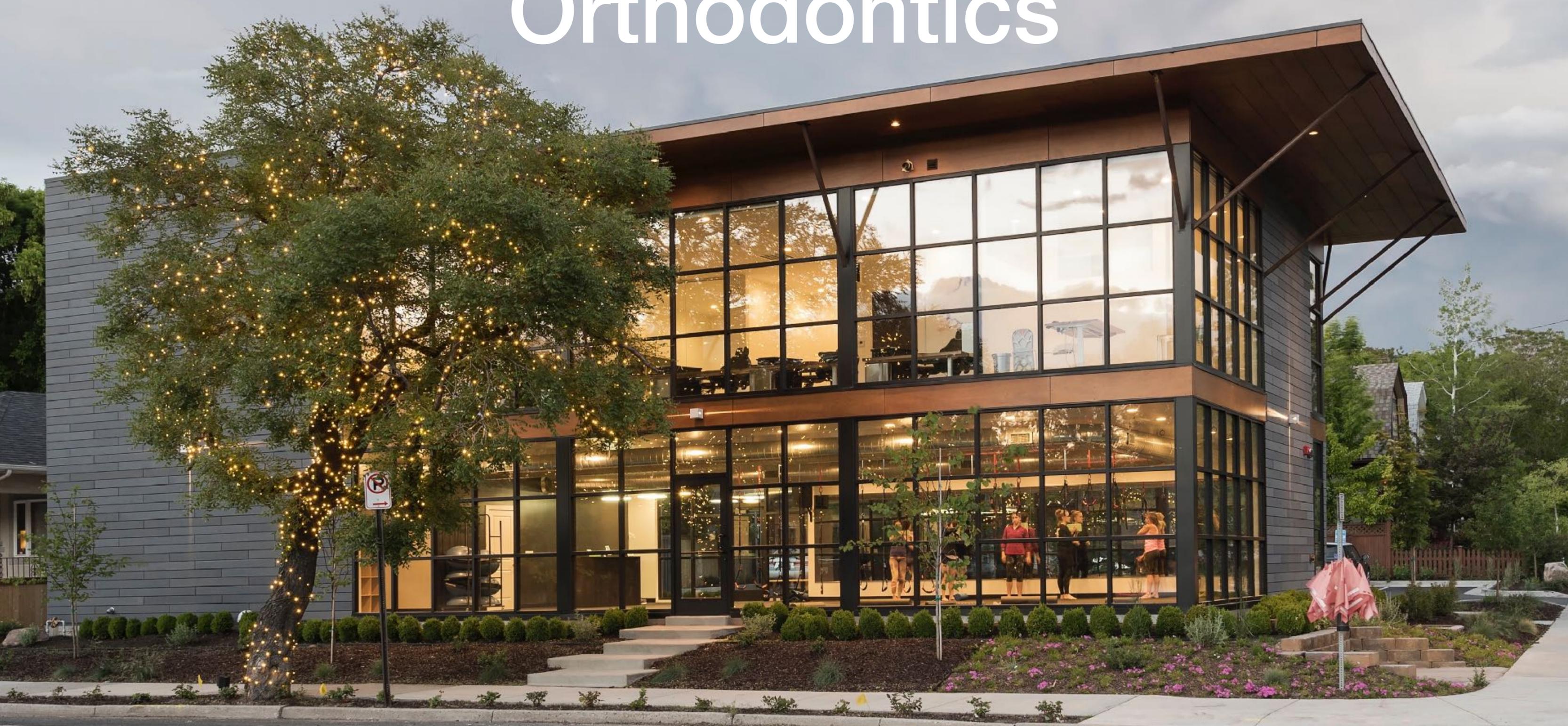
Who's Watching Our Kids?

- the orthodontist as primary child airway professional -

John Graham, DDS, MD
Salt Lake City, Utah



My Journey to Airway-Centric Orthodontics





Clinical Snapshot

- **Passive Self-Ligation**
- **Annual Extraction Rate $< 0.5\%$**
- **No Rapid Palatal Expanders**



It is difficult to free fools
from the chains they revere.

Voltaire

“ quote fancy

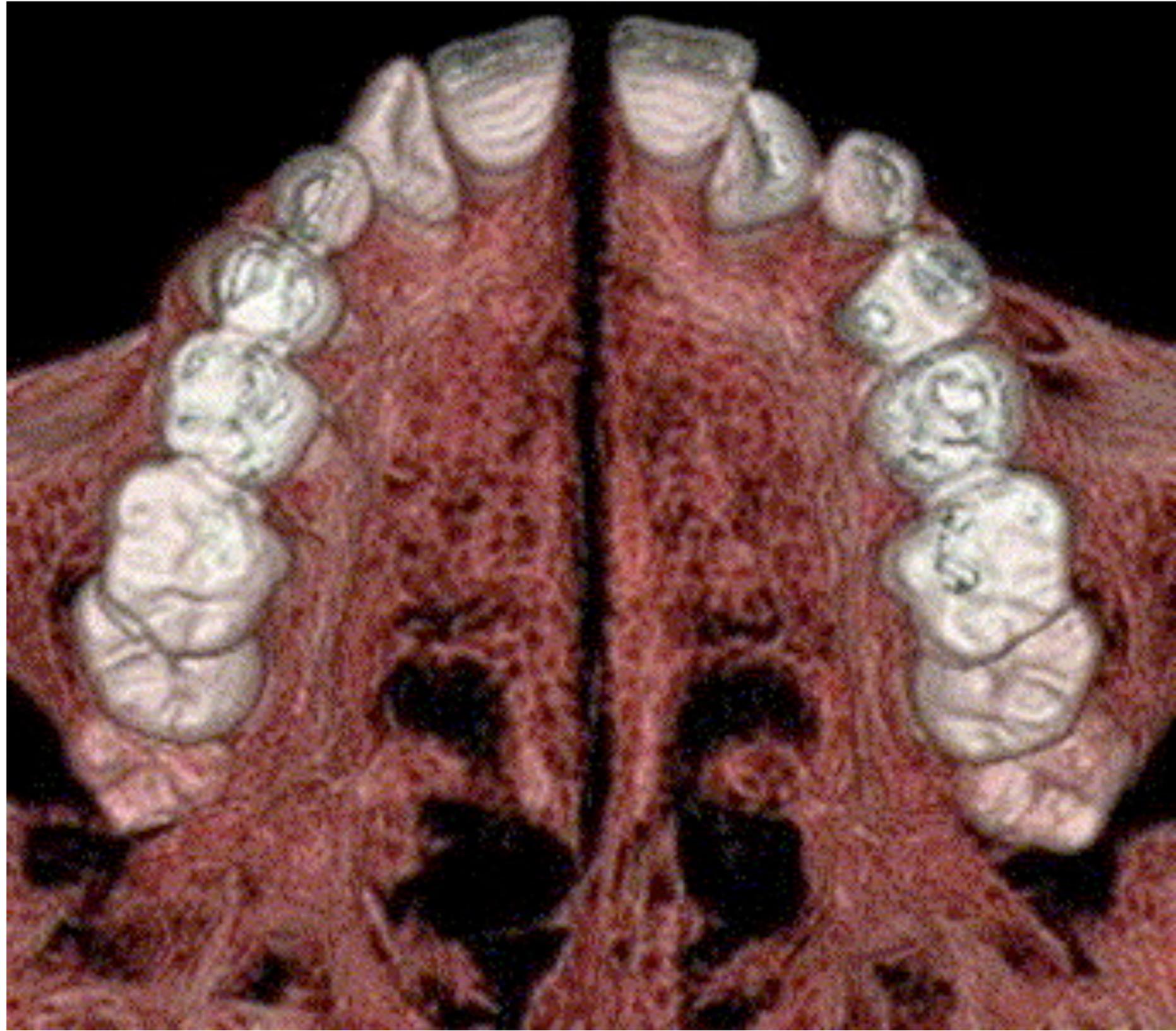
In nearly 15 years of private practice
I've never used an RPE for resolution
of crossbite or crowding.

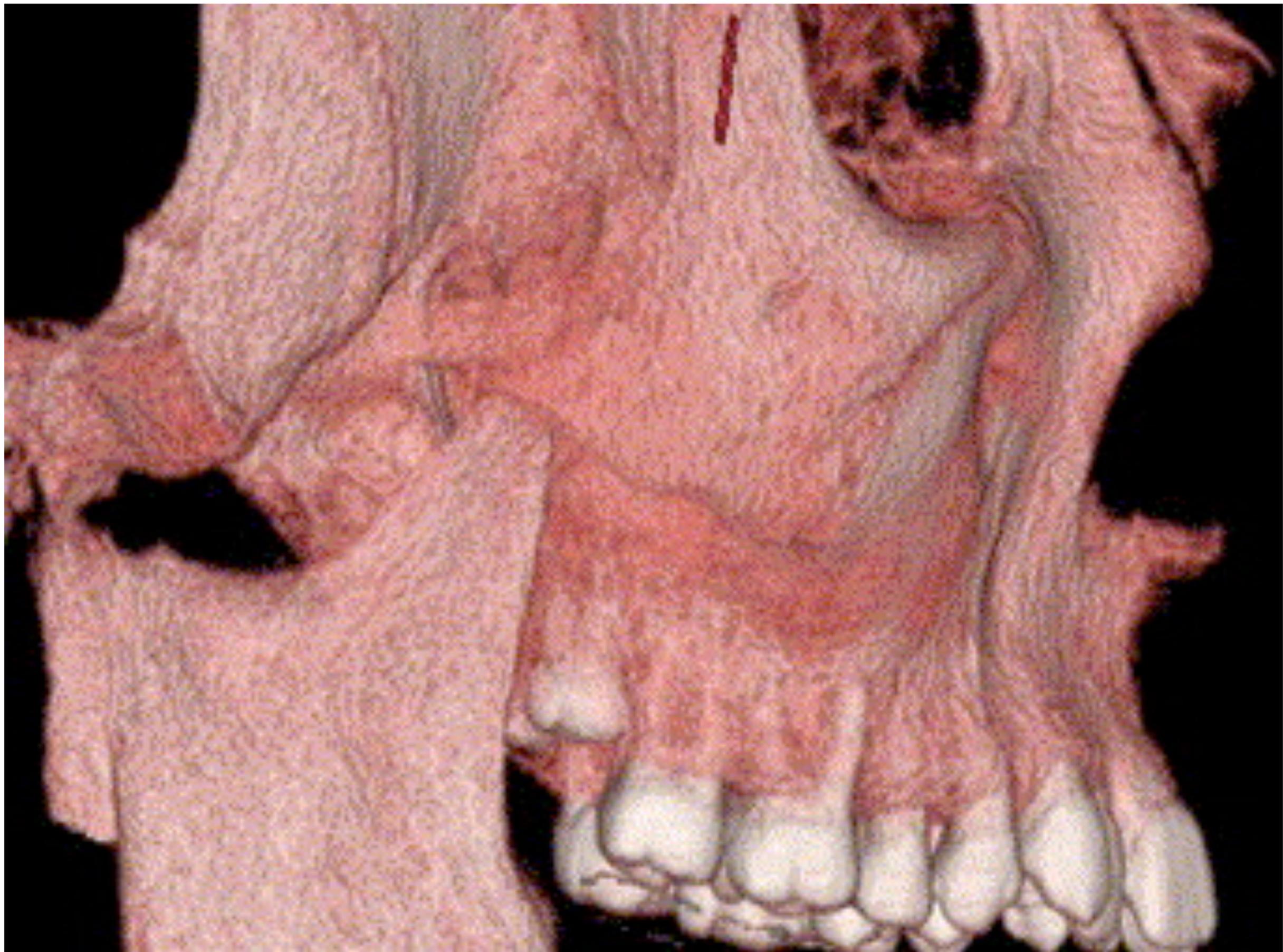
High-resolution multislice computerized tomography with multiplanar and 3-dimensional reformation imaging in rapid palatal expansion

Karin Habersack,^a Anita Karoglan,^a Bernhard Sommer,^b and Klaus U. Benner^c

Weilheim and Munich, Germany

Introduction: The purpose of this study was to evaluate whether high-resolution multislice computerized tomography (CT) with multiplanar reformation and 3-dimensional (3D) imaging is helpful in demonstrating the effects on midfacial sutures induced by rapid palatal expansion (RPE), thereby appraising and corroborating the current state of the art or possibly adding new findings. **Methods:** Two patients in different stages of skeletal maturity (aged 10 and 16 years) underwent CT examinations immediately after active opening with the RPE appliance. **Results:** The 3D CT imaging method proved to be valuable in visualizing skeletal effects on not only the midpalatal suture, but also adjacent sutures. It allowed precise 3D location of tooth positions. In the 3D CT images of 1 patient, the complete opening of the midpalatal suture was visible. The median dissection of the incisal foramen at the point of unification of the 2 nasopalatine channels could be visualized. Additionally, the positions of severely displaced maxillary canines could be located precisely. In the other patient, apart from the completely open midpalatal suture, the opening or widening of the internasal suture, the nasomaxillary sutures, and the frontomaxillary sutures were visible. **Conclusions:** The 3D imaging of high-resolution multislice CT opens up a new dimension in orofacial diagnosis. Improvements of the quantity and the exactness of diagnostic parameters were attained. The imaging method is helpful and indicated in RPE patients with additional diagnostic objectives related to the development of occlusion. This imaging method is recommended in borderline cases (juvenile or adult patients with questionable sutural response) to determine whether the suture is completely open or whether surgical support is needed. (*Am J Orthod Dentofacial Orthop* 2007;131:776-81)







COMP: Class I, Invisalign

12	to	12	Status	Consultation
			Recall	
02/21/18			Brackets	
			Bal \$	0.00
			Ttl Chr	0 hrs - 41 min
			Ttl Dr	0:18:08
			Birth Date	9/5/81
			City	Holladay

of 12-12 Target to

Chart Note P. Prob. Ch. Compl. Dispos.

Last Entry

U AW

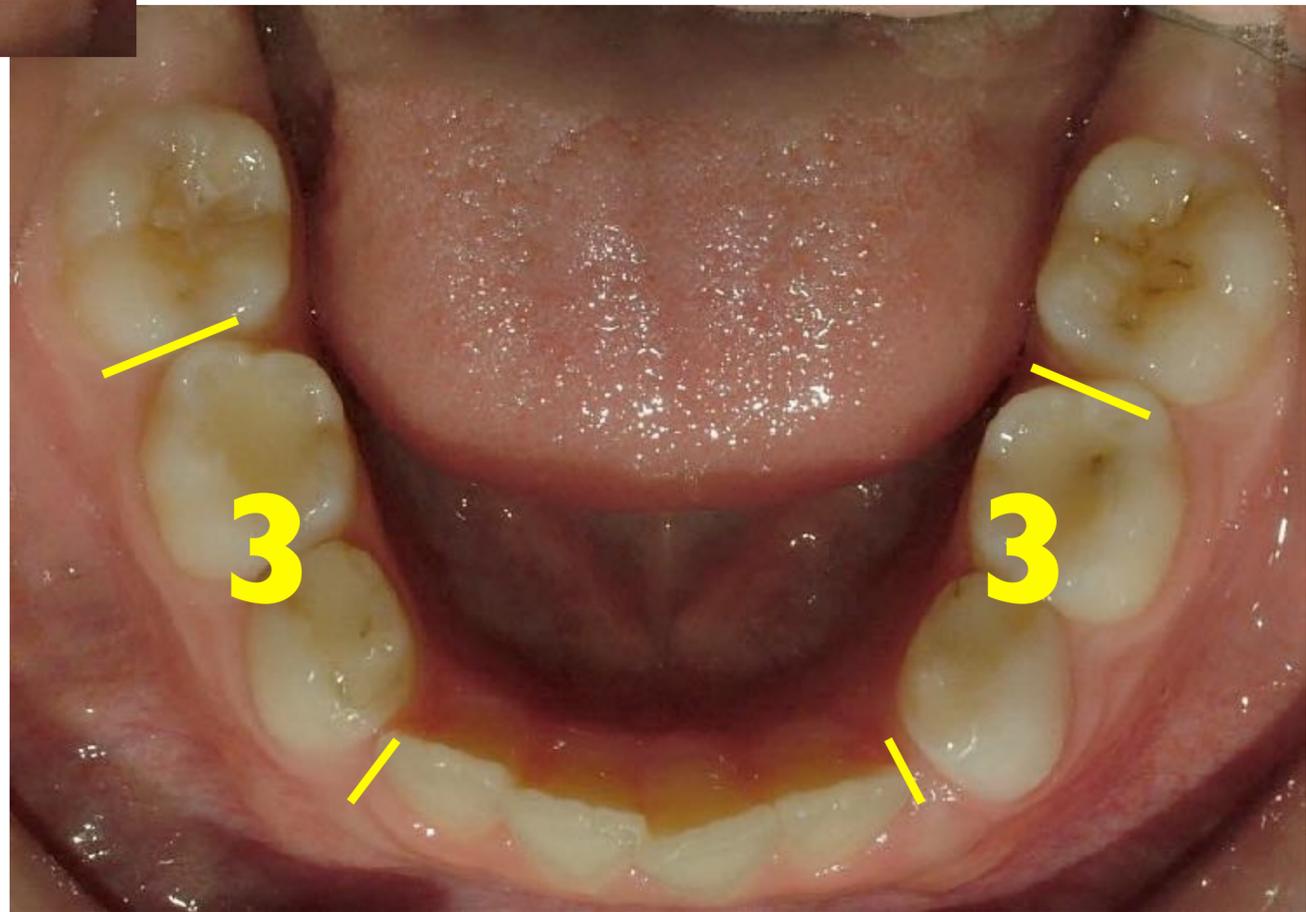
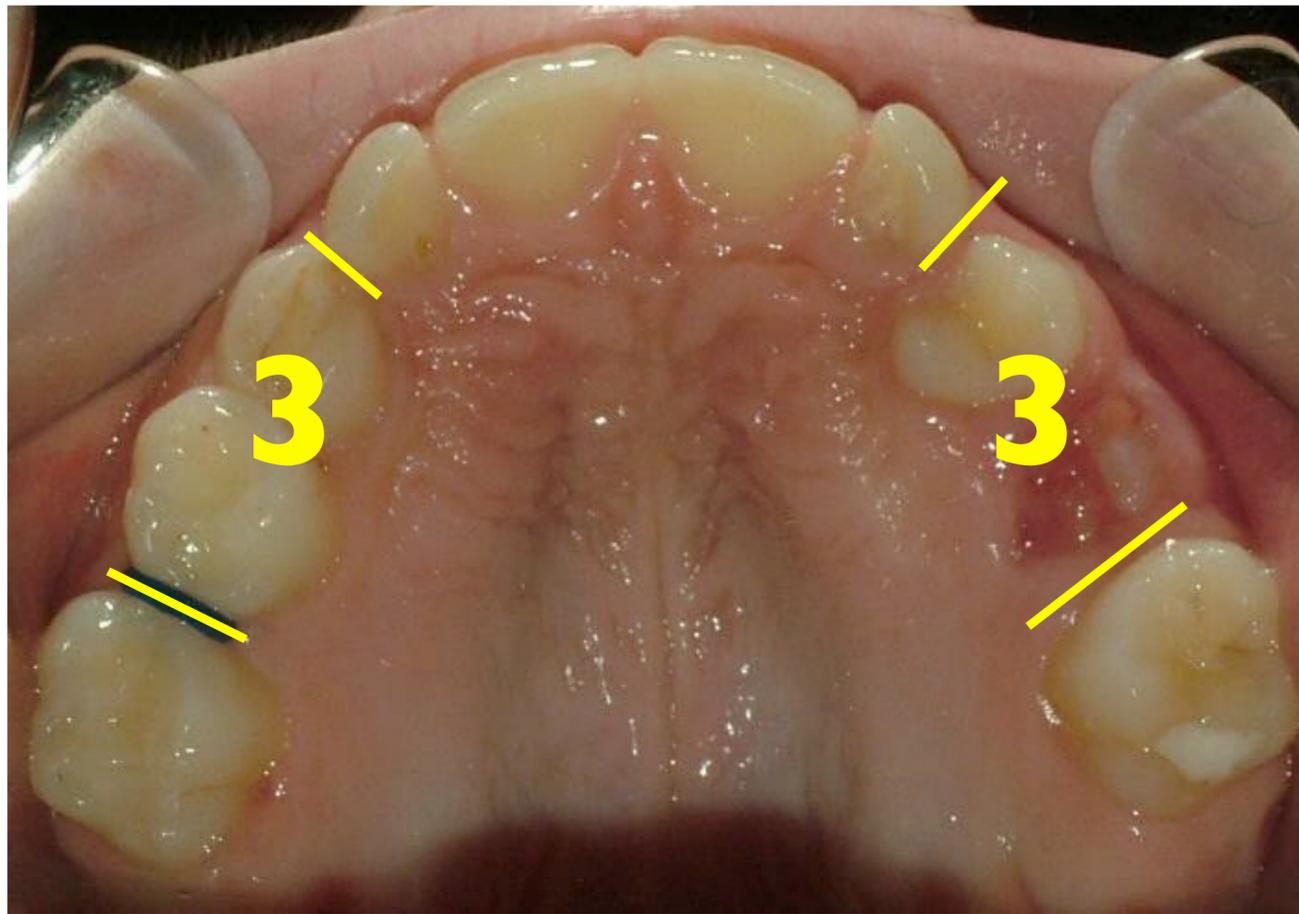
Phase: Unassigned Show All Sort by: Treatment Pt Notes

Date	As.	Dr.	Type	Today's Procedure	Proc Code	XRay	OJ/OB	AW's	Elastics/Hrs	Lo	OH A/E	AP	PR	Next	
02/21/18	CG	JW G	V	Consult: CT Scan, Photos, Exam. Had braces and an expander in Jr High/HS. No retainers given. Teeth are shifting and leaning in towards tongue. Dr G recommends comp full tx w/Invisalign. D1, 8-12 months to open up bite and fix crowding. Went over attachments, IPR, refinement scans and possible elastic wear.										B	contract invisali

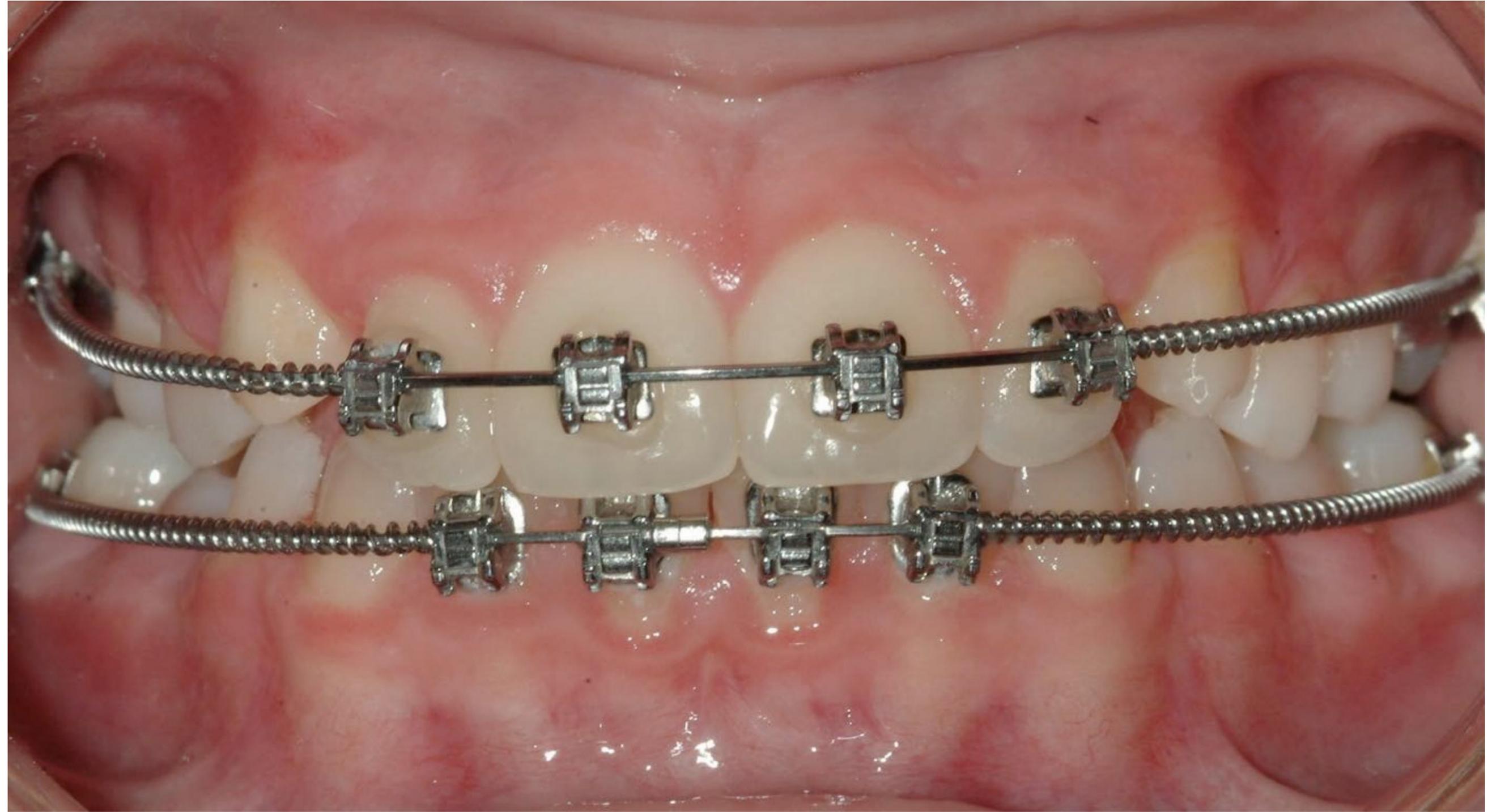


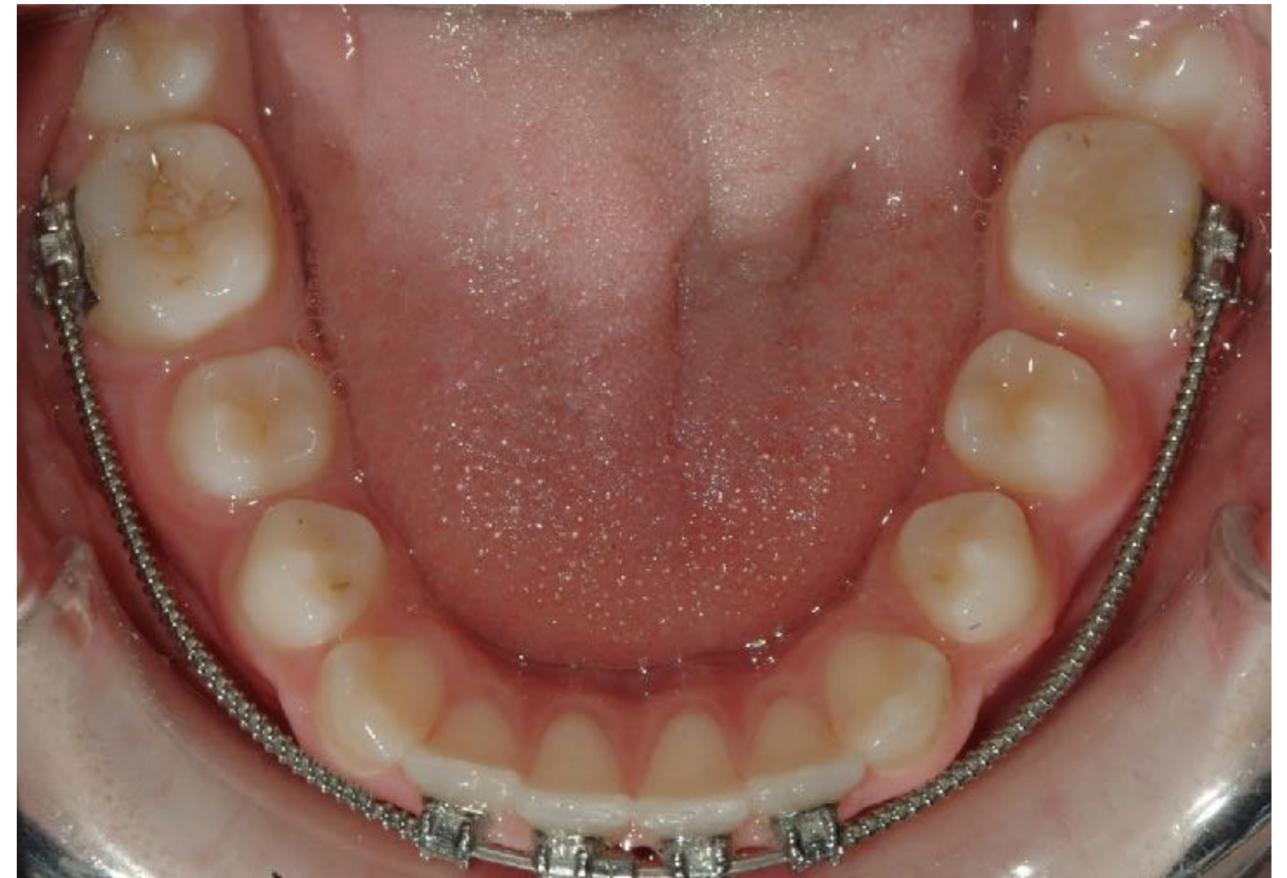
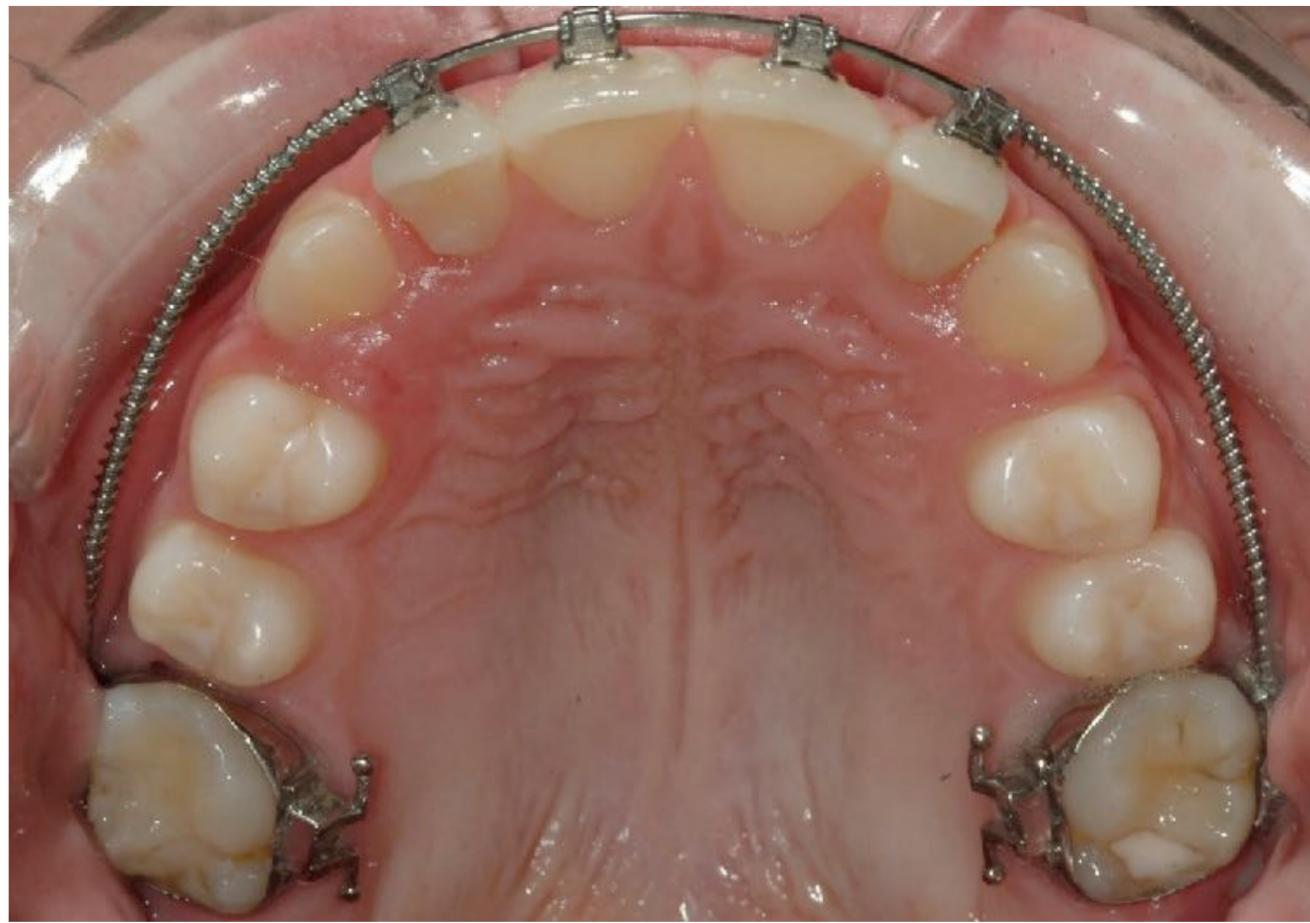
**Severe Crowding
Resolved Without RPEs Or Extractions**

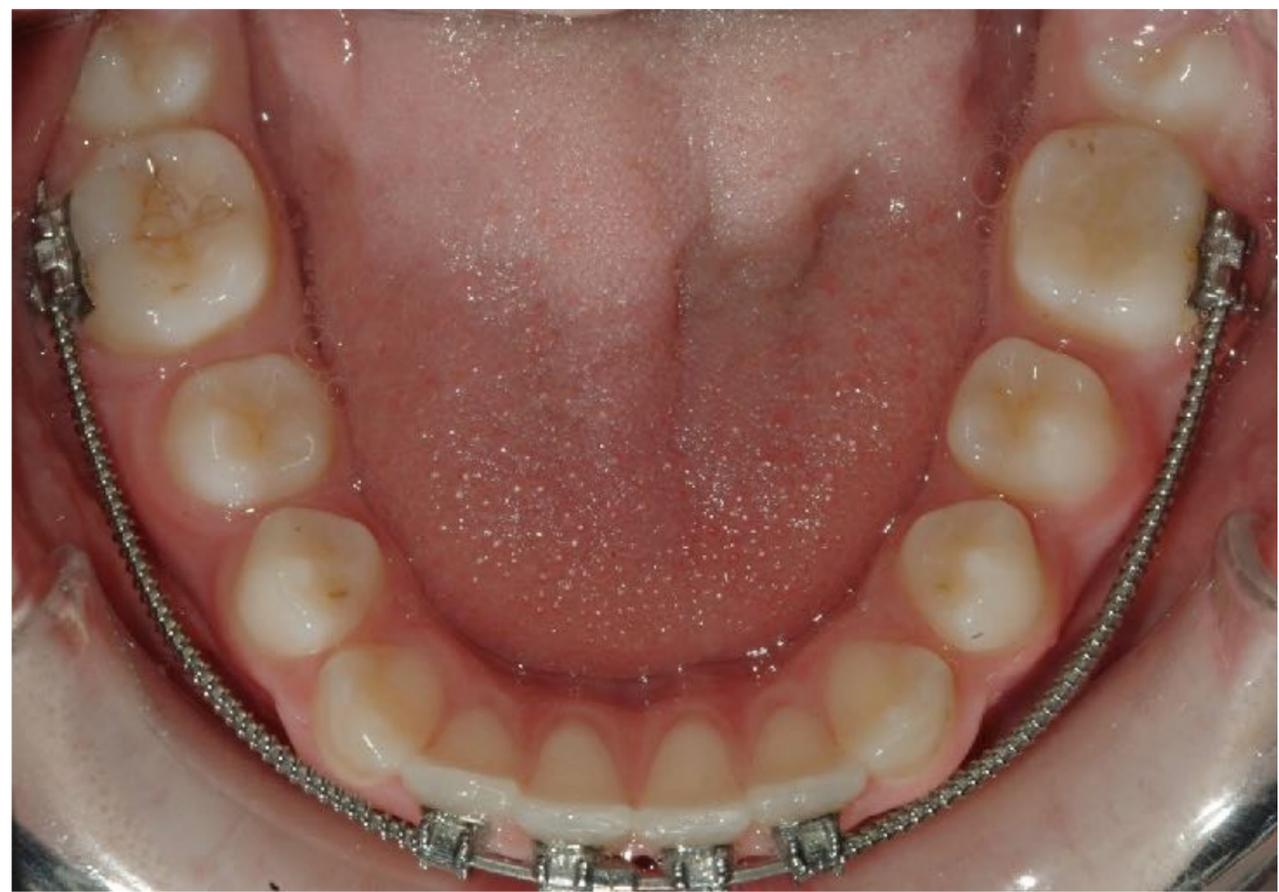
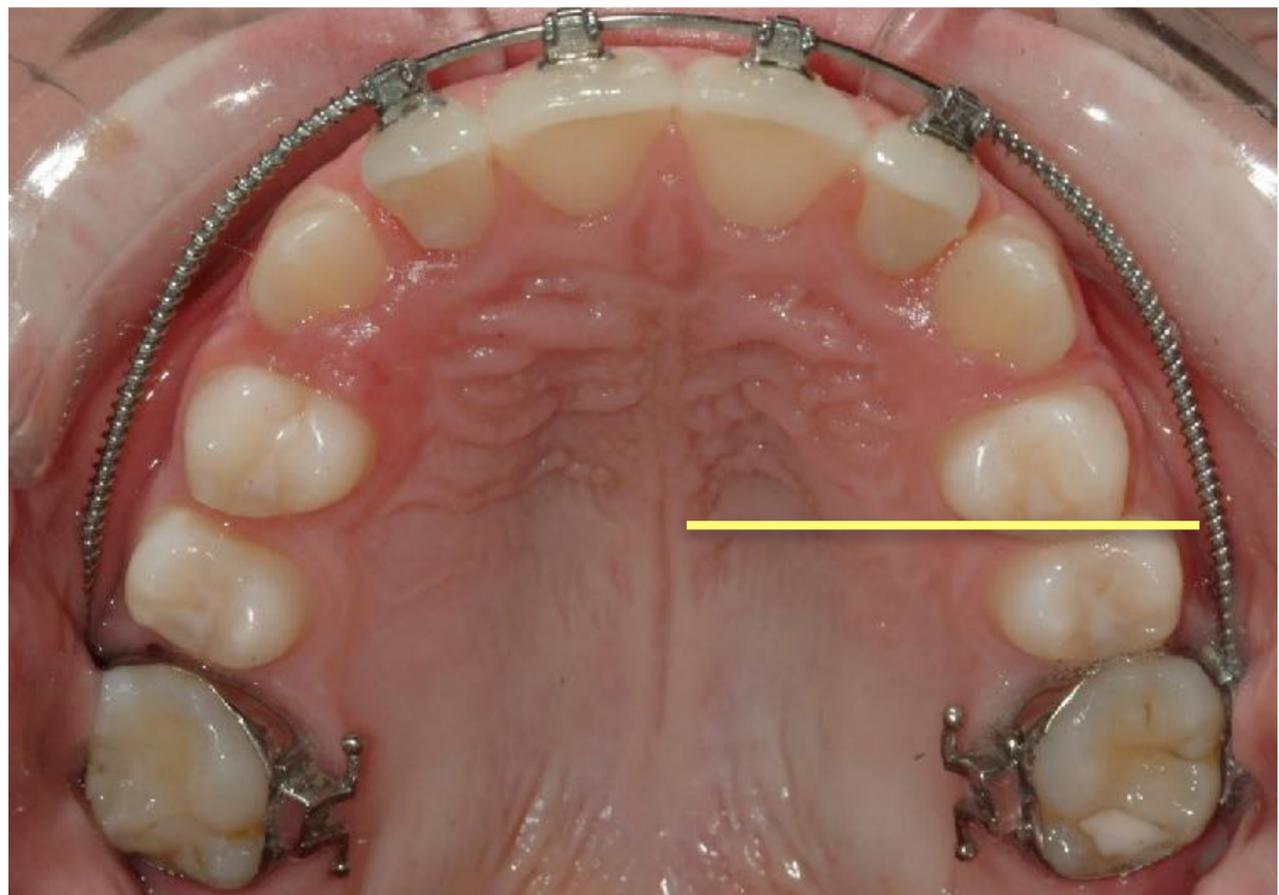
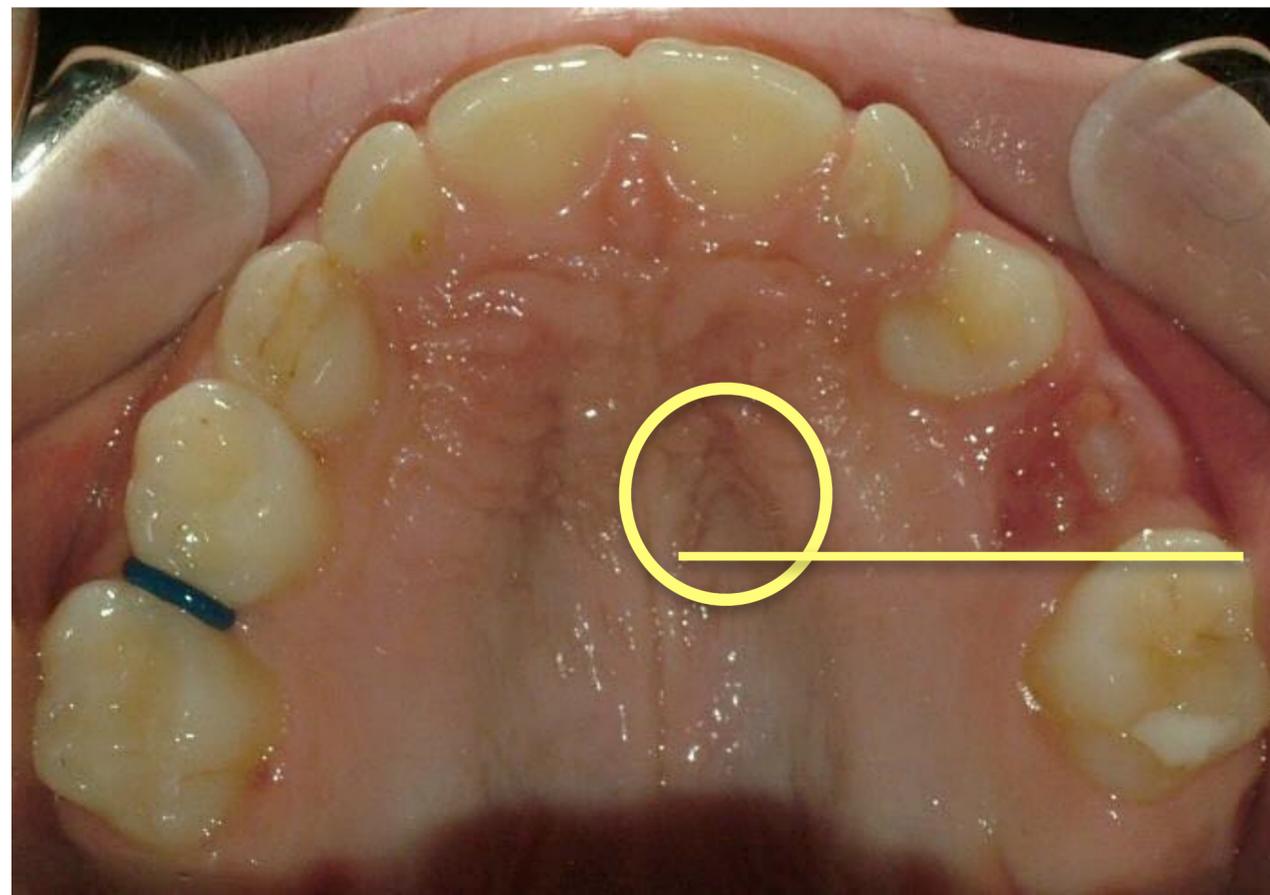


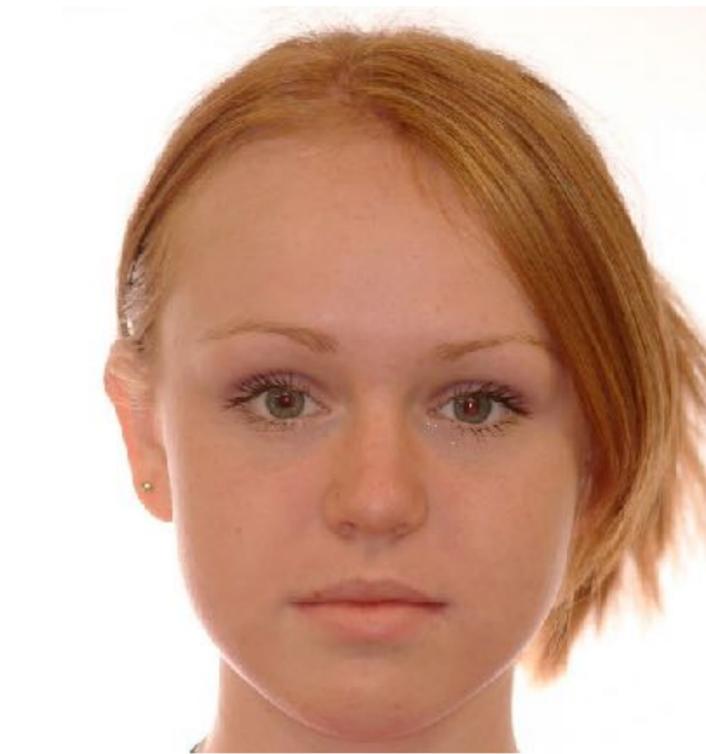


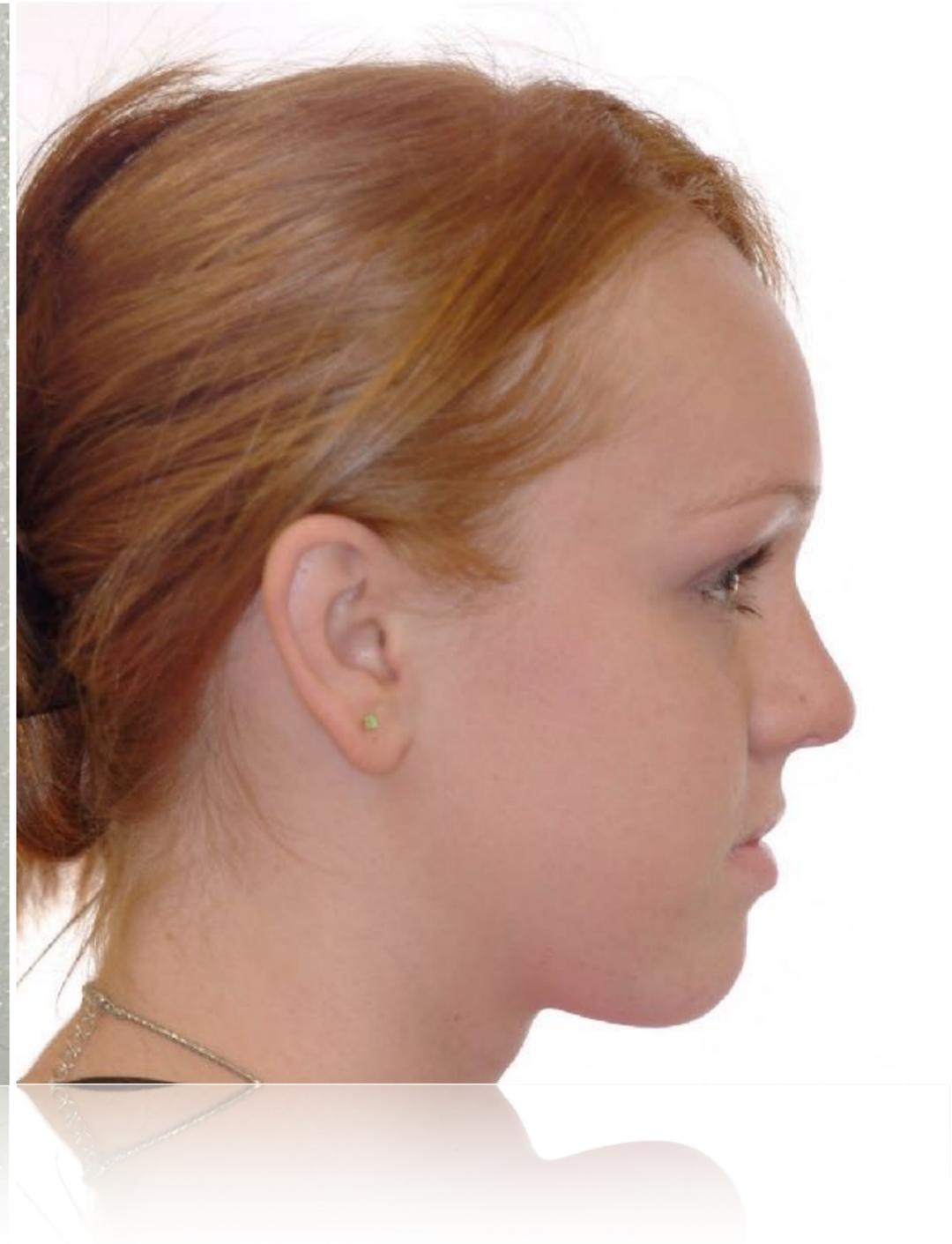




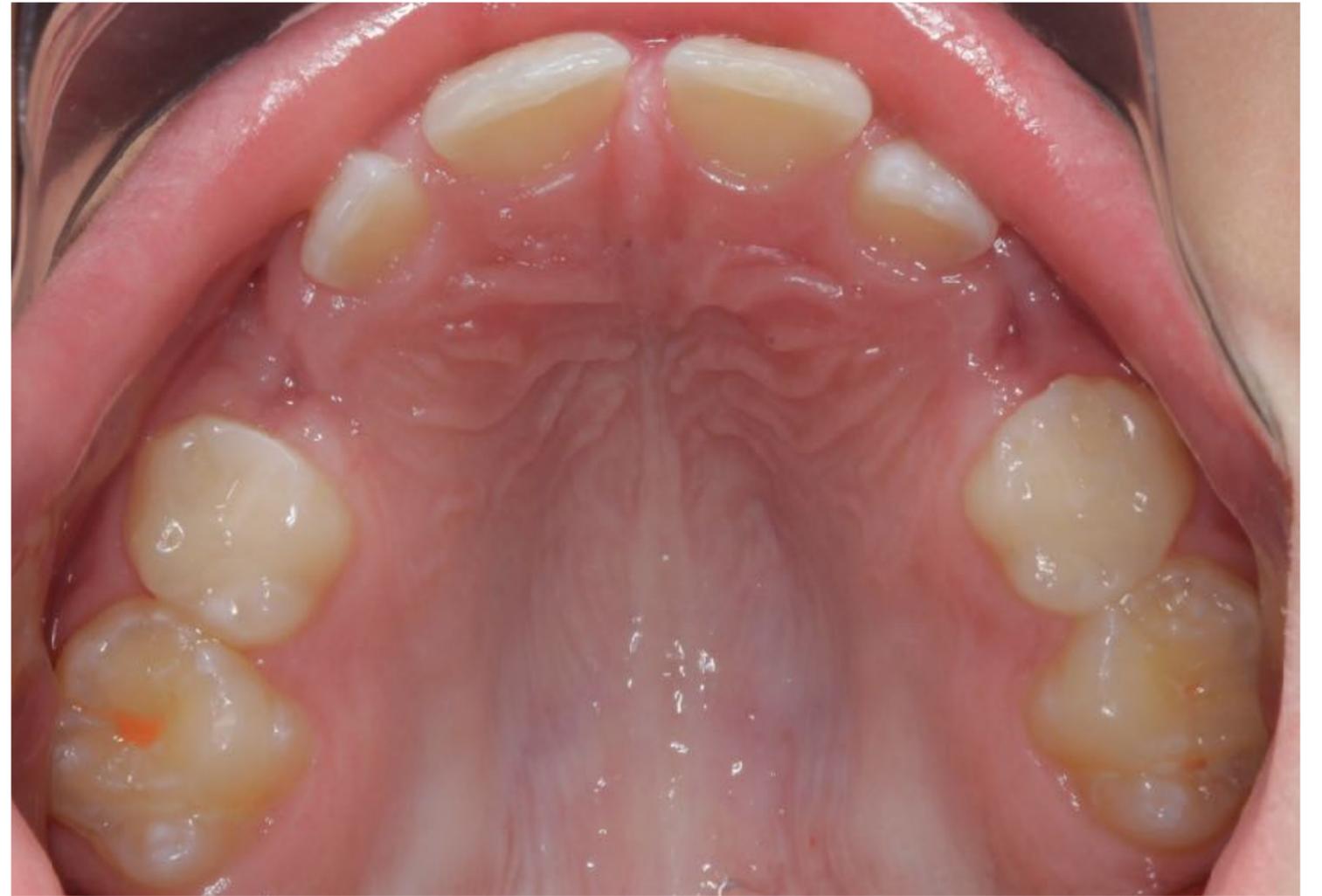














Parents told he needed Phase I *after*
four bicuspids were extracted

Is there a place in my
practice for rapid palatal
expanders?

Yes...

Yes...

and it loosened the
chains I revered

It took a decade, an iCAT,
and my daughter to figure
it out



**Taylor, the day of
her
adenotonsillectomy**

Obstructive Sleep Apnea (OSA)

Nocturnal enuresis decreased significantly in all patients after the RME period, and all patients showed full dryness after 3 years[†]

Nocturnal enuresis ceased within a few months in the 10 cases studied by using rapid maxillary expansion to reduce nasal constriction^{††}

Surgical removal of upper airway obstruction led to a significant decrease in or complete cure of nocturnal enuresis in 76% of children studied^{*}

[†]Timms D, Rapid maxillary expansion in the treatment of nocturnal enuresis, Angle Orthodontist 1990, 60(3):229-33

^{††}Namer Al-Taal, et al, Effect of rapid maxillary expansion on monosymptomatic primary NE , Angle Orthodontist 2015, 85(1):102-108

^{*}Weider, DJ, Nocturnal enuresis in children with upper airway obstruction, Otolaryngol Head Neck Surg 1991;105:417-32

Obstructive Sleep Apnea (OSA)

OSA is one of the most common causes of sleep-disordered breathing in the general pediatric population*

Reported prevalence ranging from 1-5% *†

* Bixler EO, Vgontzas AN, Lin HM, Liao D, Calhoun S, Vela-Bueno A, et al. Sleep disordered breathing in children in a general population sample: prevalence and risk factors. *Sleep*. 2009;32(6):731–6

† Rosen CL, Larkin EK, Kirchner HL, Emancipator JL, Bivins SF, Surovec SA, et al. Prevalence and risk factors for sleep-disordered breathing in 8 to 11-year-old children: association with race and prematurity. *J Pediatr*. 2003;142(4):383–9.

Obstructive Sleep Apnea (OSA)

- Peak prevalence occurs at 2-8 years
- Symptoms are very nonspecific and require a high level of suspicion
- Daytime symptoms may include some or all:
 - Difficulty concentrating
 - Morning headaches
 - Daytime sleepiness
 - ADHD

Obstructive Sleep Apnea (OSA)

25% of children with ADHD snore.

Of those 81% could have their ADHD
eliminated if their habitual snoring were
effectively treated*

*Chervin, RD et al. Symptoms of Sleep Disorders, Inattention, and Hyperactivity in Children, 1997, Sleep, 20(12):1185-1192.



Treatment Outcomes of Adenotonsillectomy in Children with Obstructive Sleep Apnea: A Prospective Longitudinal Study

Yu-Shu Huang, MD^{1,5}; Christian Guilleminault, DM, MD, DBiol^{2,4}; Li-Ang Lee, MD³

¹Department of Child Psychiatry and Sleep Center, Chang Gung Memorial Hospital, Taoyuan, Taiwan; ²Department of Otolaryngology at Stanford University, Stanford, CA; ³Department of Otolaryngology at National Sun Yat-sen University, Kaohsiung, Taiwan; ⁴Department of Cranio Facial Center and Sleep Center, National Sun Yat-sen University, Kaohsiung, Taiwan; ⁵Department of Education, National Chia-Yi University, Chiayi, Taiwan; ⁶University, Taipei, Taiwan

Objective: To evaluate the efficacy of adenotonsillectomy (AT) in the treatment of obstructive sleep apnea (OSA) in children.

Study Design: An investigation of children (6 to 12 y old) with OSA and obstructive sleep apnea (OSA) was conducted using polysomnography, examination, questionnaires, and polysomnography.

Multivariate generalized linear modeling and hierarchical linear modeling were used for analysis of OSA, and Generalized Linear Models were used for analysis of OSA.

Results: Of the 135 children, 88 terminated the study at 36 months (boys: 8.9 ± 2.04 y, girls: 8.8 ± 2.07 y; body mass index [BMI] = 19.1 ± 1.35 and a mean postoperative AHI at 6 mo (AHI₆) of 3.47 ± 1.8 in AHI was noted with a mean AHI₃₆ = 6.48 ± 5.57 events/h and AHI₃₆ > 5 in the OSA-18 questionnaire.

The residual pediatric OSA after AT was significantly associated with BMI, and recurrence of pediatric OSA was significantly associated with enurges and rate of change in BMI and body weight.

Conclusions: Adenotonsillectomy leads to significant improvement in OSA, but worsening over time was observed in 68% of our cases.

Keywords: adenotonsillectomy, comorbidity, obstructive sleep apnea, polysomnography

Citation: Huang YS; Guilleminault C; Lee LA; Lin CH; Hwang FM. Treatment outcomes of adenotonsillectomy in children with obstructive sleep apnea: a prospective longitudinal study. *SLEEP* 2014;37(1):71-76.

INTRODUCTION

Obstructive sleep apnea (OSA) syndrome is a highly prevalent condition in children and characterized by snoring, witnessed apnea, unrefreshing sleep, and excessive daytime sleepiness.^{1,2} Children with OSA experience recurrent periods of elevated upper airway resistance during sleep due to partial or complete upper airway obstruction, which results in snoring, episodic oxyhemoglobin desaturation, hypercapnia, and repeated arousals.^{3,4} The respiratory disturbance of recurrent hypoxia-reoxygenation episodes during the night is associated with an increased risk of suboptimal growth, poor sleep quality, neurocognitive dysfunction, behavioral problems, overweight status, and cardiovascular disease in childhood.⁵⁻⁸ The prevalence of OSA is approximately 2-3% in children,^{9,10} and current studies have evaluated the influence of OSA on various associated morbidities^{5-8,11} and tried to identify the factors predicting poor treatment outcome.¹²

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Address correspondence to: Christian Guilleminault, DM, MD, DBiol, Stanford University Sleep Medicine Division, 450 Broadway Street, MC 5704, Redwood City, CA 94063; Tel: (650) 723-6601; E-mail: cguil@stanford.edu

Orthodontic Expansion Treatment and Adenotonsillectomy in the Treatment of Obstructive Sleep Apnea in Prepubertal Children

Christian Guilleminault, MD, BiolD¹; Stacey Quo, DDS²; Nelly T. Huynh, PhD¹; Kasey Li, MD, DDS¹

¹Stanford University Sleep Medicine Program, Stanford, CA; ²University of California San Francisco School of Dentistry, San Francisco, CA

Study objective: Rapid maxillary expansion and adenotonsillectomy are proven treatments of obstructive sleep apnea (OSA) in children. Our goal was to investigate whether rapid maxillary expansion should be offered as an alternative to surgery in select patients. In addition, if both therapies are required, the order in which to perform these interventions needs to be determined.

Design: Prepubertal children with moderate OSA clinically judged to require both adenotonsillectomy and orthodontic treatment were randomized into 2 treatment groups. Group 1 underwent adenotonsillectomy followed by orthodontic expansion. Group 2 underwent orthodontic expansion followed by adenotonsillectomy.

Subjects: Thirty-two children (16 girls) in an academic sleep clinic.

Method: Clinical evaluation and polysomnography were performed after each stage to assess efficacy of each treatment modality.

Results: The 2 groups were similar in age, symptoms, apnea-hypopnea index, and lowest oxygen saturation. Two children with orthodontic treatment first did not require subsequent adenotonsillectomy. Thirty

children underwent both treatments. Two of them were still symptomatic and presented with abnormal polysomnogram results following therapies. In the remaining 28 children, all results were significantly different from those at entry ($P = 0.001$) and from single therapy ($P = 0.01$), regardless of the order of treatment. Both therapies were necessary to obtain complete resolution of OSA.

Conclusion: In our study, 87.5% of the children with sleep-disordered breathing had both treatments. In terms of treatment order, 28 children underwent orthodontic treatment alone, whereas no child underwent surgery alone to resolve OSA. Two children who underwent both treatments continued to have OSA.

Keywords: Prepubertal children, pediatric obstructive sleep apnea, orthodontics, adenotonsillectomy, treatment

Citation: Guilleminault C; Quo S; Huynh NT; Li K. Orthodontic expansion treatment and adenotonsillectomy in the treatment of obstructive sleep apnea in prepubertal children. *SLEEP* 2008;31(7):953-959.

THE IMPACT OF RAPID MAXILLARY EXPANSION (RME) ON NASAL RESISTANCE AND NOCTURNAL SLEEP SYMPTOMS IN CHILDREN WAS DESCRIBED even before the syndrome of obstructive sleep apnea syndrome (OSAS) in children.¹⁻⁶ RME has been used to treat children presenting with OSAS.⁷⁻⁹ In these studies, children either had adenotonsillectomy prior to orthodontic treatment or had an isolated problem involving the maxilla. However, most children seen in sleep clinics present with moderate to large tonsils, scored between 2+ and 4+ on the Friedman et al scale,¹⁰ and a narrow upper airway with maxillary constriction and/or some degree of mandibular retrusion, which presents as a narrow and long face.¹¹⁻¹⁴ There are unresolved questions in this commonly seen child. Should orthodontic treatment be considered first or should adenotonsillectomy be performed as first-line treatment? Can orthodontic expansion be an isolated treatment option for a child with OSAS who still has adenoids and tonsils? Also, while it is known that the persistence of tonsillar enlargement may interfere with orthodontic treatment,

Disclosure Statement

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Accepted for publication March, 2008

Address correspondence to: Christian Guilleminault, MD, BiolD, Stanford University Sleep Disorders Clinic, 401 Quarry Rd suite 3301, Stanford, CA, 94305; Tel: (650) 723-6601; Fax: (650) 725-0910; E-mail: cguil@stanford.edu

no data is available on the impact of combined therapies in children with OSAS.

In our clinical practice, we educate families that adenotonsillectomy and orthodontic expansion may be necessary. The order of these treatments has largely been a decision of convenience or parental preference, influenced, for example, by appointment availability or timing of school vacation. In this study, prepubertal children with moderate OSAS as a lowest oxygen saturation of 90% and an apnea-hypopnea index [AHI] of 20 events per hour) needing both adenotonsillectomy and orthodontic treatment were prospectively randomized into 2 treatment groups. Each treatment group underwent baseline polysomnography recording. Group 1 was randomized to undergo adenotonsillectomy followed by orthodontic expansion (maxillary or bimaxillary expansion). Group 2 was randomized to undergo the reverse sequence, starting with orthodontic expansion, followed by adenotonsillectomy. Polysomnography was performed after each stage to assess the efficacy of each treatment modality.

METHODS

All the children studied were referred to the Stanford University Sleep Disorders Clinic for symptoms known to be associated with obstructive sleep apnea. They were seen in a team setting and evaluated by an otolaryngologist, an orthodontist, and a certified sleep physician. Each child underwent a comprehensive assessment, including the Pediatric Sleep Questionnaire, a general pediatric physical examination, and a sleep study. Oral examination was performed to assess tonsil and tongue position with respect to the position

Tongue posture improvement and pharyngeal airway enlargement as secondary effects of rapid maxillary expansion: A cone-beam computed tomography study

Tomonori Iwasaki,^a Issei Saitoh,^b Yoshihiko Takemoto,^c Emi Inada,^c Eriko Kakuno,^d Ryuzo Kanomi,^d Haruaki Hayasaki,^e and Youichi Yamasaki^f
Kagoshima, Niigata, and Himeji, Japan

Introduction: Rapid maxillary expansion (RME) is known to improve nasal airway ventilation. Recent evidence suggests that RME is an effective treatment for obstructive sleep apnea in children with maxillary constriction. However, the effect of RME on tongue posture and pharyngeal airway volume in children with nasal airway obstruction is not clear. In this study, we evaluated these effects using cone-beam computed tomography. **Methods:** Twenty-eight treatment subjects (mean age 9.96 ± 1.21 years) who required RME treatment had cone-beam computed tomography images taken before and after RME. Twenty control subjects (mean age 9.68 ± 1.02 years) received regular orthodontic treatment. Nasal airway ventilation was analyzed by using computational fluid dynamics, and intraoral airway (the low tongue space between tongue and palate) and pharyngeal airway volumes were measured. **Results:** Intraoral airway volume decreased significantly in the RME group from 1212.9 ± 1370.9 mm³ before RME to 279.7 ± 472.0 mm³ after RME. Nasal airway ventilation was significantly correlated with intraoral airway volume. The increase of pharyngeal airway volume in the control group (1226.3 ± 1782.5 mm³) was only 41% that of the RME group (3015.4 ± 1297.6 mm³). **Conclusions:** In children with nasal obstruction, RME not only reduces nasal obstruction but also raises tongue posture and enlarges the pharyngeal airway. (*Am J Orthod Dentofacial Orthop* 2013;143:235-45)

^aLecturer, Field of Developmental Medicine, Health Research Course, Graduate School of Medical and Dental Sciences, Kagoshima University, Kagoshima, Japan.

^bAssociate professor, Division of Pediatric Dentistry, Department of Oral Health Science, Course of Oral Life Science, Graduate School of Medical and Dental Sciences, Niigata University, Niigata, Japan.

^cResearch associate, Field of Developmental Medicine, Health Research Course, Graduate School of Medical and Dental Sciences, Kagoshima University, Kagoshima, Japan.

^dPrivate practice, Himeji, Japan.

^eProfessor and chairman, Division of Pediatric Dentistry, Department of Oral Health Science, Course of Oral Life Science, Graduate School of Medical and Dental Sciences, Niigata University, Niigata, Japan.

^fProfessor and chairman, Field of Developmental Medicine, Health Research Course, Graduate School of Medical and Dental Sciences, Kagoshima University, Kagoshima, Japan.

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Reprint requests to: Tomonori Iwasaki, Graduate School of Medical and Dental Sciences, Kagoshima University, 8-35-1, Sakuragaoka Kagoshima-City, Kagoshima, 890-8544, Japan; e-mail: yamasaki@dent.kagoshima-u.ac.jp.

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Nasal breathing allows proper growth and development of the craniofacial complex. In contrast, nasal obstruction that leads to mouth breathing results in lower tongue posture (with greater intraoral airway volume) and a constricted and V-shaped maxillary dental arch.¹

Rapid maxillary expansion (RME) has been widely used by orthodontists to increase the maxillary transverse dimensions of young patients. Recent studies have suggested that RME also increases nasal width and volume.²⁻⁵ Therefore, RME is generally thought to diminish the resistance to nasal airflow.^{6,7} Gray⁸ investigated the medial results of RME in 310 patients and found that over 80% of them changed their breathing pattern from mouth breathing to nasal breathing. Furthermore, the efficacy of RME to treat obstructive sleep apnea syndrome (OSAS) in children has been reported.⁹⁻¹¹ However, the mechanism behind the RME effect is not clear. OSAS in children has various causes.¹² Our purpose was to clarify a mechanism by which RME improves the symptoms.

Upper airway obstruction has also been associated with low tongue posture; among its other effects, RME

Rapid maxillary expansion and obstructive sleep apnea: A review and meta-analysis

Almiro-José Machado-Júnior ¹, Edilson Zancanella ², Agrício-Nubiato Crespo ³

¹ DDS, PhD. Discipline of Otorhinolaryngology, Unicamp (Campinas State University) - São Paulo - Brazil

² MD, PhD. Discipline of Otorhinolaryngology, Unicamp (Campinas State University) - São Paulo - Brazil

³ MD, PhD (Full professor). Discipline of Otorhinolaryngology, Unicamp (Campinas State University) - São Paulo - Brazil

Correspondence:

Rua Maria Monteiro, 841 ap 11 Cambuí,

13025-151 Campinas,

551932535472SP - Brazil,

almirom@ig.com.br

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Rapid maxillary expansion and obstructive sleep apnea: A review and meta-analysis

Table 1. Selected studies to conduct the meta-analysis.

Author	Year	N	Male	Female	Age	AHI 0	AHI 1	Follow-up	p-value	Evidence level
Guilleminault <i>et al.</i> (19)	2011	15			6.5+-0.2SEM	11.1+-0.7	5.4+-0.6	3	0.15	2B
Villa <i>et al.</i> (20)	2011	10			6.6+-2.1 SD	6.3+-4.7	2.4+-2.0	12	0.05	3B
Miano <i>et al.</i> (21)	2009	9	6	3	6,4+-1.97 SD	17.4+-21	5.4+-6.25	12	0.02	3B
Villa <i>et al.</i> (22)	2007	16	9	7	6.6+-2.0 SD	5.8+-6.8	1.5+-1.6	12	0.005	3B
Pirelli <i>et al.</i> (23)	2004	31	19	12	8.7	12.18+-2.6	0.4+-1.1	4	0.0001	3B
Pirelli <i>et al.</i> (24)	2005	42	26	16	7.3	12.17+-2.5	0.5+-1.2	4	0.000	3B
Pirelli <i>et al.</i> (25)	2012	40				12.8	6.5+-3.1	4	0.05	3B
Villa <i>et al.</i> (26)	2013	22			8.20+-2.62 SD	5.81±6.05	2.64±3.11	12	0.005	3B
Caprioglio <i>et al.</i> (27)	2013	14			7.1+-0.6 SD	5.7+-1.2	1.4+-0.6	12	<0.001	3B
Guilleminault <i>et al.</i> (28)	2008	16		16	6.45+-0.8SD	12.2+-4.0	5.1+-3.8	6		3B

Rapid maxillary expansion and obstructive sleep apnea: A review and meta-analysis

- 10 studies met inclusion criteria (215 children)
- Non-syndromic children between 0 and 12 years
- Diagnosis of OSAS from polysomnography who underwent RME with polysomnography after
- AHI available before and after RME

Rapid maxillary expansion and obstructive sleep apnea: A review and meta-analysis

Meta-analysis conclusions

- There is a decrease in AHI after RME in children with OSAS
- AHI decline is maintained as indicated by follow-up tests ranging from 3 months to 14 years
- RME in children with OSAS appears to be another effective treatment

Minimum Cross-sectional Area (MCA) As A Screening tool for OSA Risk

High risk OSA = 0-52 mm²
Mod risk OSA = 52-110 mm²
Low risk OSA > 110 mm²

Consentini T, Le Donne R, Mancini D, et al: Magnetic resonance imaging of the upper airway in obstructive sleep apnea. Radiol Med 108:404, 2004

Strelzow VV, Banks RHI, Basile A, et al: Cephalometric airway analysis in obstructive sleep apnea syndrome. Laryngoscope 98:1149, 1988

Lowe AA, Gionhaku N, Takeuchi K, et al. Three dimensional CT reconstructions of tongue and airway in adult subjects with obstructive sleep apnea. Am J Orthod Dentofacial Orthop 1986; 90(5):364-74.

Avrahami E, Englender M. Relation between CT axial cross-sectional area of the oropharynx and obstructive sleep apnea syndrome in adults. AJNR Am J Neuroradiol 1995;16(1):135-40.

Ogawa T, Enciso R, Shintaku WH, et al. Evaluation of cross-section airway configuration of obstructive sleep apnea. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103(1):102-8.

Poiseuille's Law

Vessel resistance (R) is directly proportional to the length (L) of the vessel and the viscosity (η) of the blood, and inversely proportional to the radius to the fourth power (r^4).

$$R \propto \frac{\eta L}{r^4}$$

Caroline



OSA Risk
High Risk = 0-52 mm²
Moderate Risk = 52-110 mm²
Low Risk > 110 mm²

MCA = 63.6 mm²

Clipping: 41%
Level/Brightness: 500 / 0.00
Window/Contrast: 3000 / 0.00

txSTUDIO

100.0

150.0

200.0

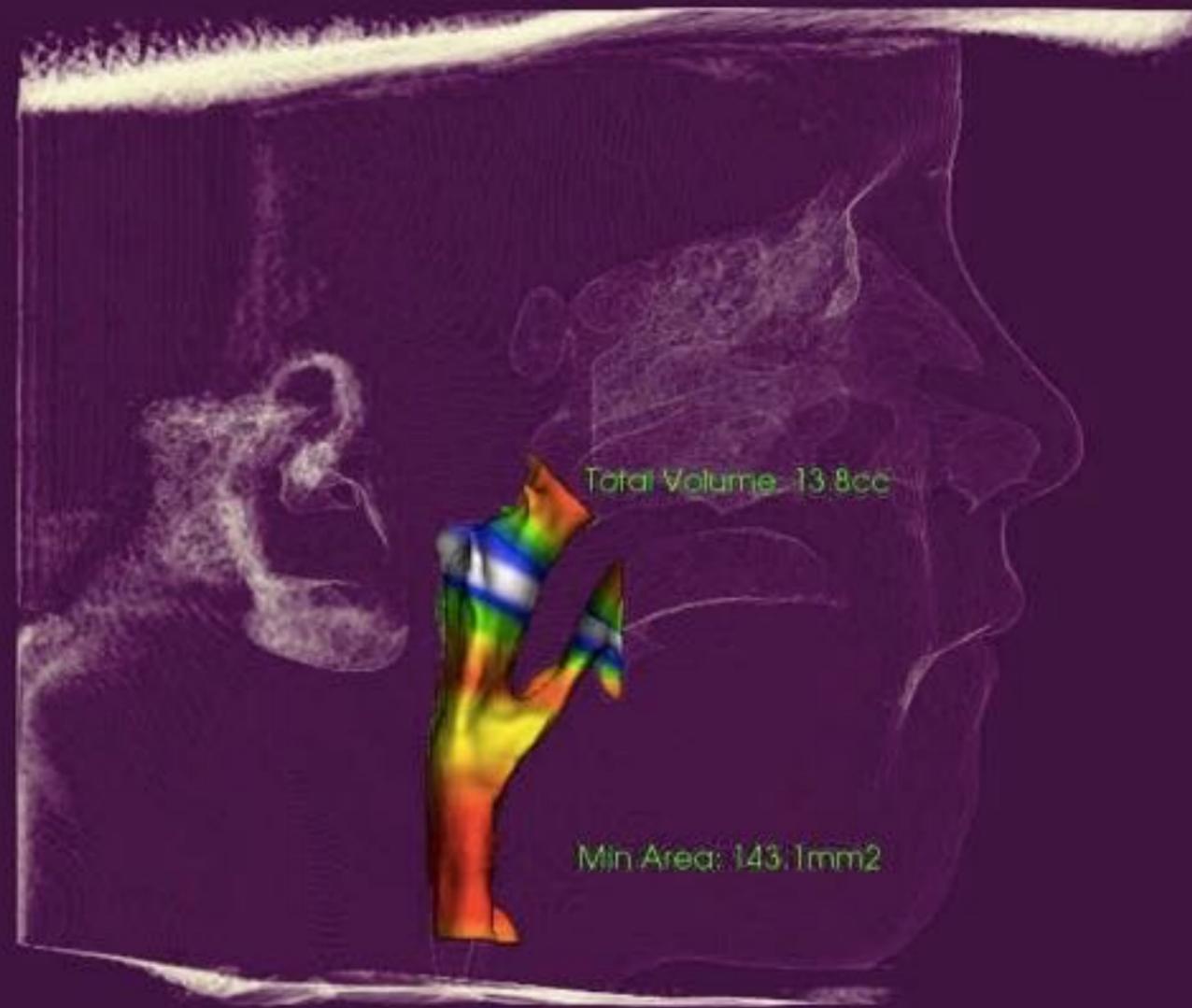
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400.0

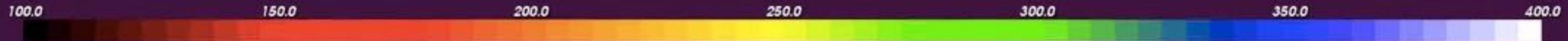
Caroline One Year Later



OSA Risk
High Risk = 0-52 mm²
Moderate Risk = 52-110 mm²
Low Risk > 110 mm²

MCA = 143.1 mm²

Clipping: 48%
Level/Brightness: 500 / 0.00
Window/Contrast: 3000 / 0.00

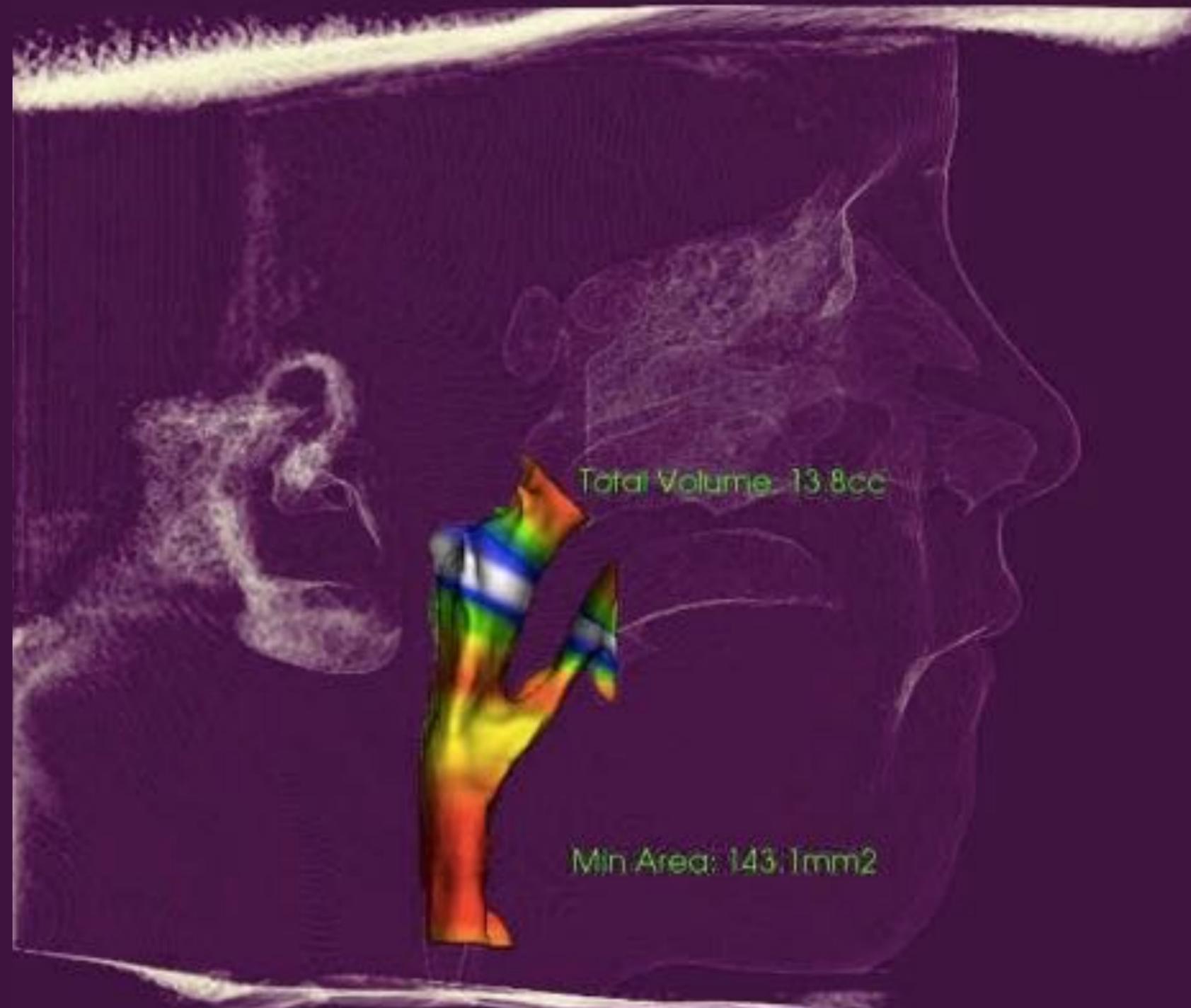


TxSTUDIO

No Treatment



MCA = 63.6 mm²



MCA = 143.1 mm²





MCA = 63.6 mm²



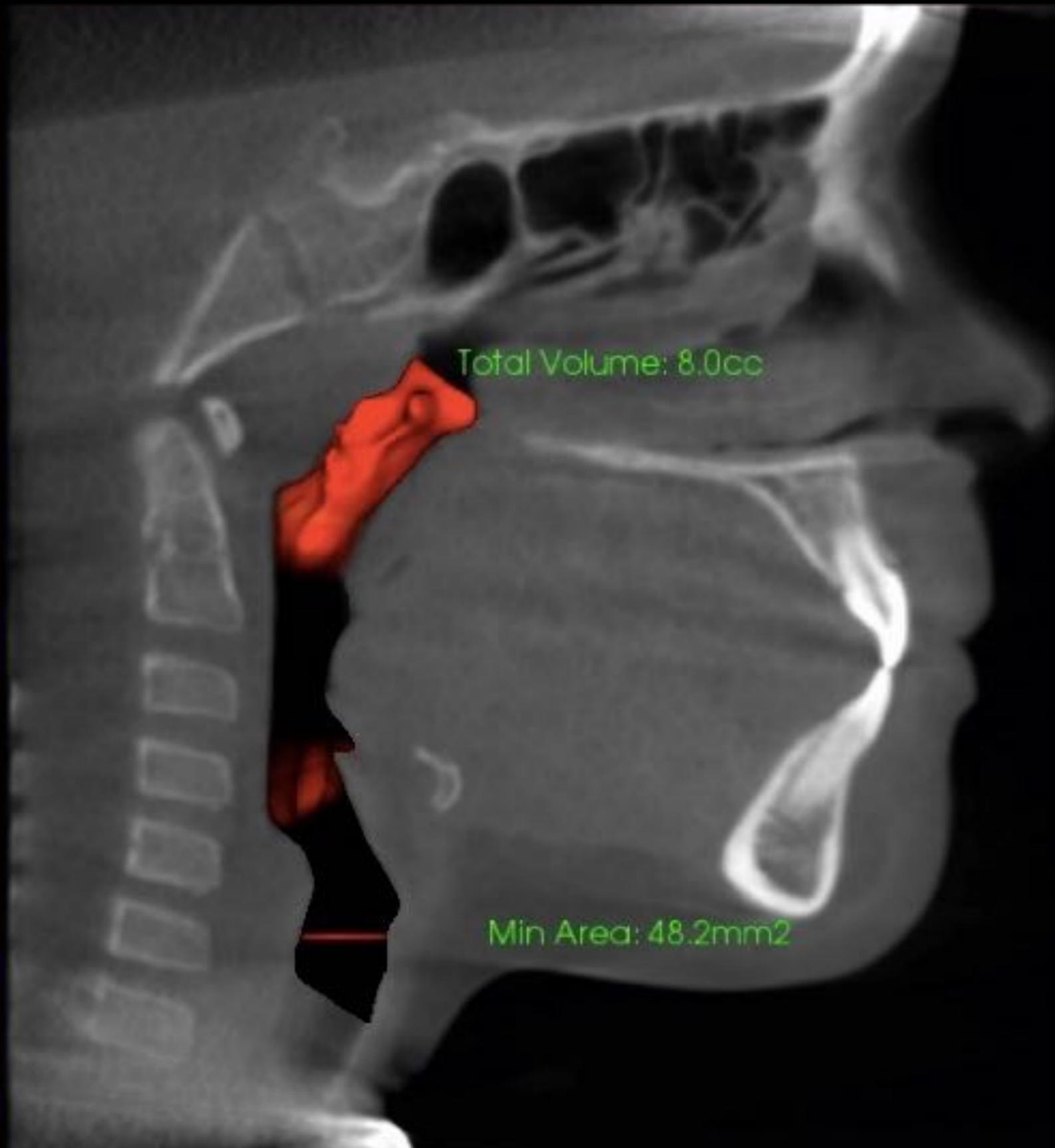
MCA = 143.1 mm²

OSA As A Family Trait

OSA in Families



Olivia 9.6



OSA Risk
High Risk = 0-52 mm²
Moderate Risk = 52-110 mm²
Low Risk > 110 mm²

MCA = 48.2 mm²

OSA in Families



Wyatt 37.12



OSA Risk
High Risk = 0-52 mm²
Moderate Risk = 52-110 mm²
Low Risk > 110 mm²

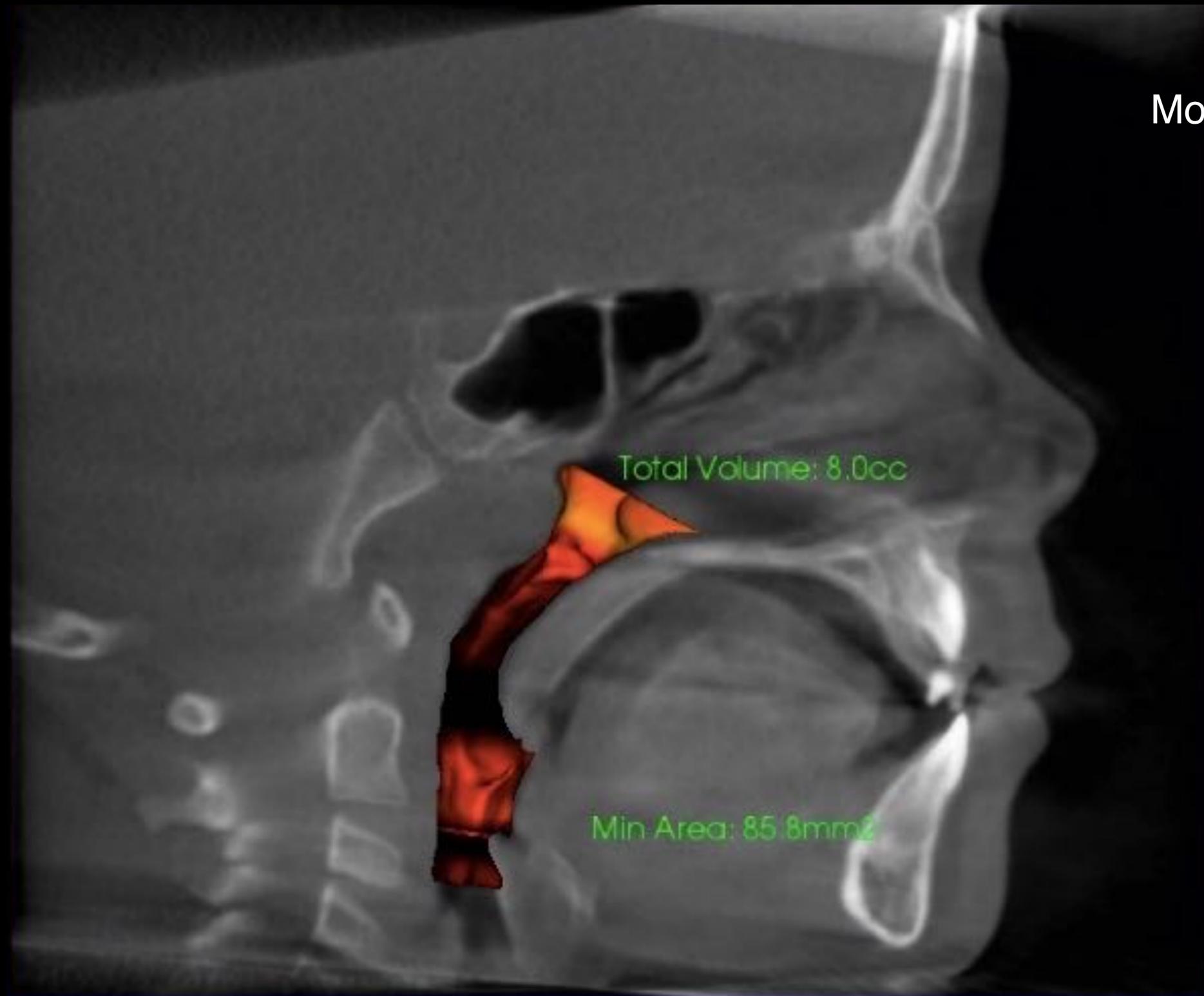


MCA = 43.5 mm²

OSA in Families



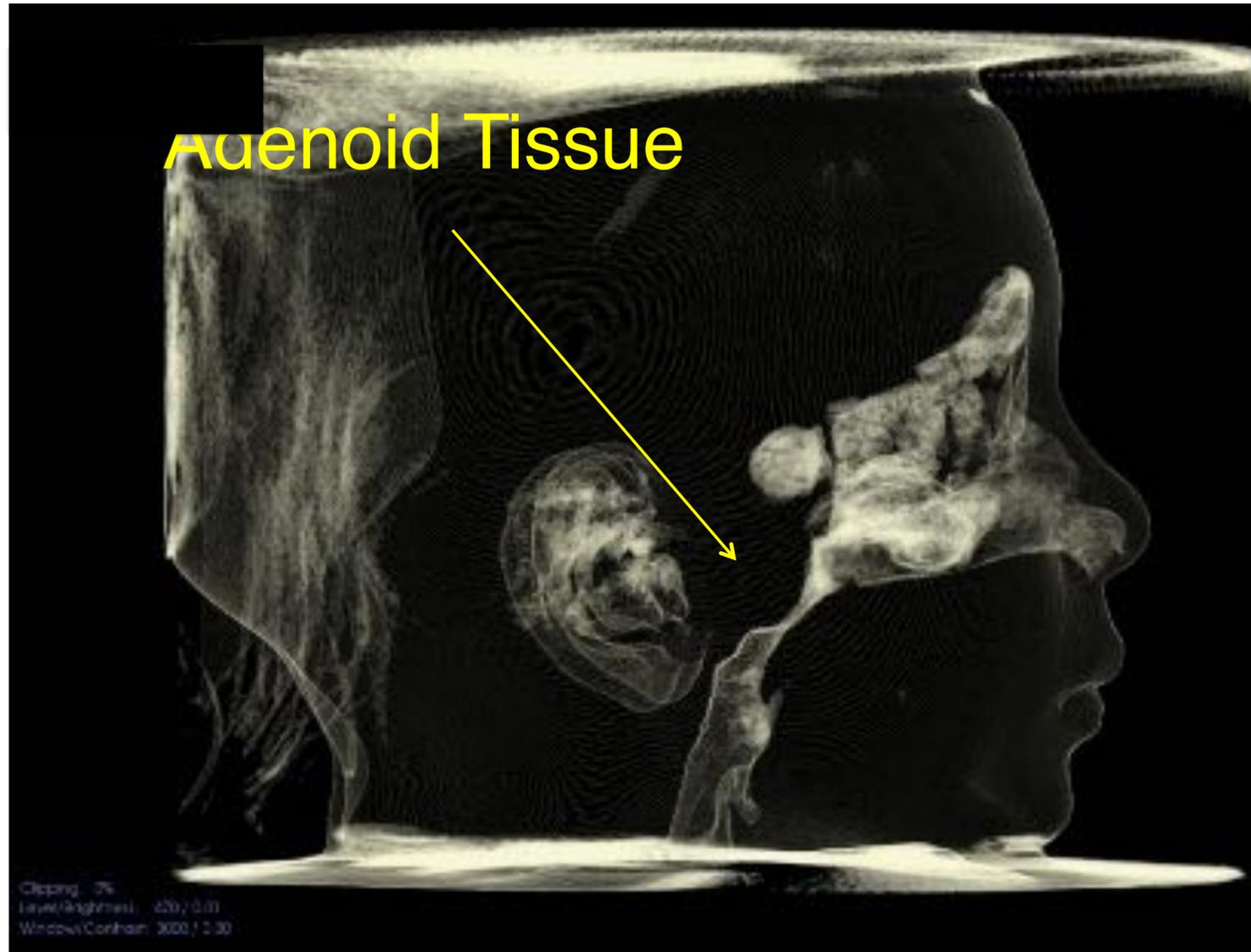
Calvin - 7.9



OSA Risk
High Risk = 0-52 mm²
Moderate Risk = 52-110 mm²
Low Risk > 110 mm²

MCA = 85.8 mm²

2 Year Airway Difference



Pre-treatment MCA = 23.8 mm²

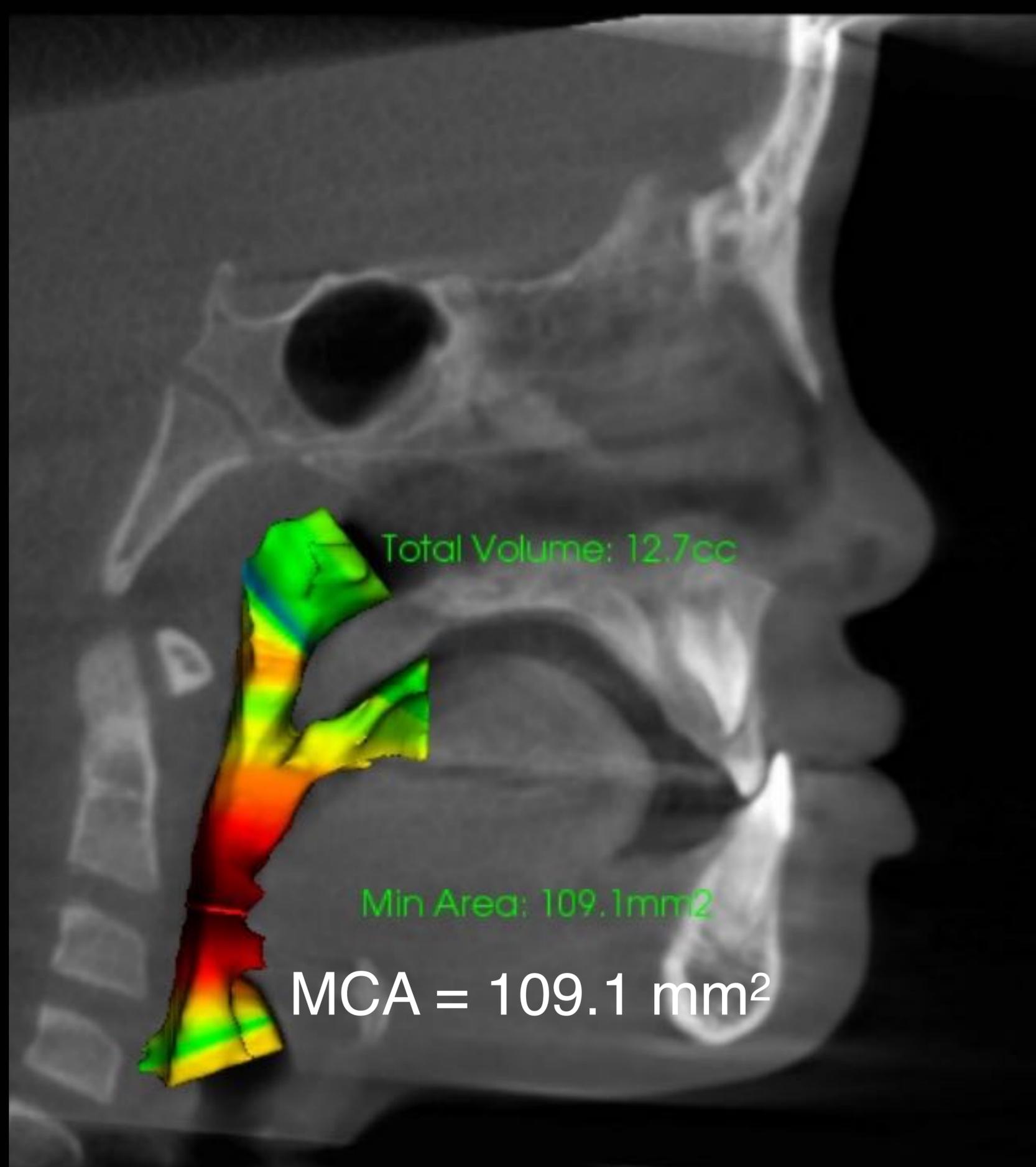


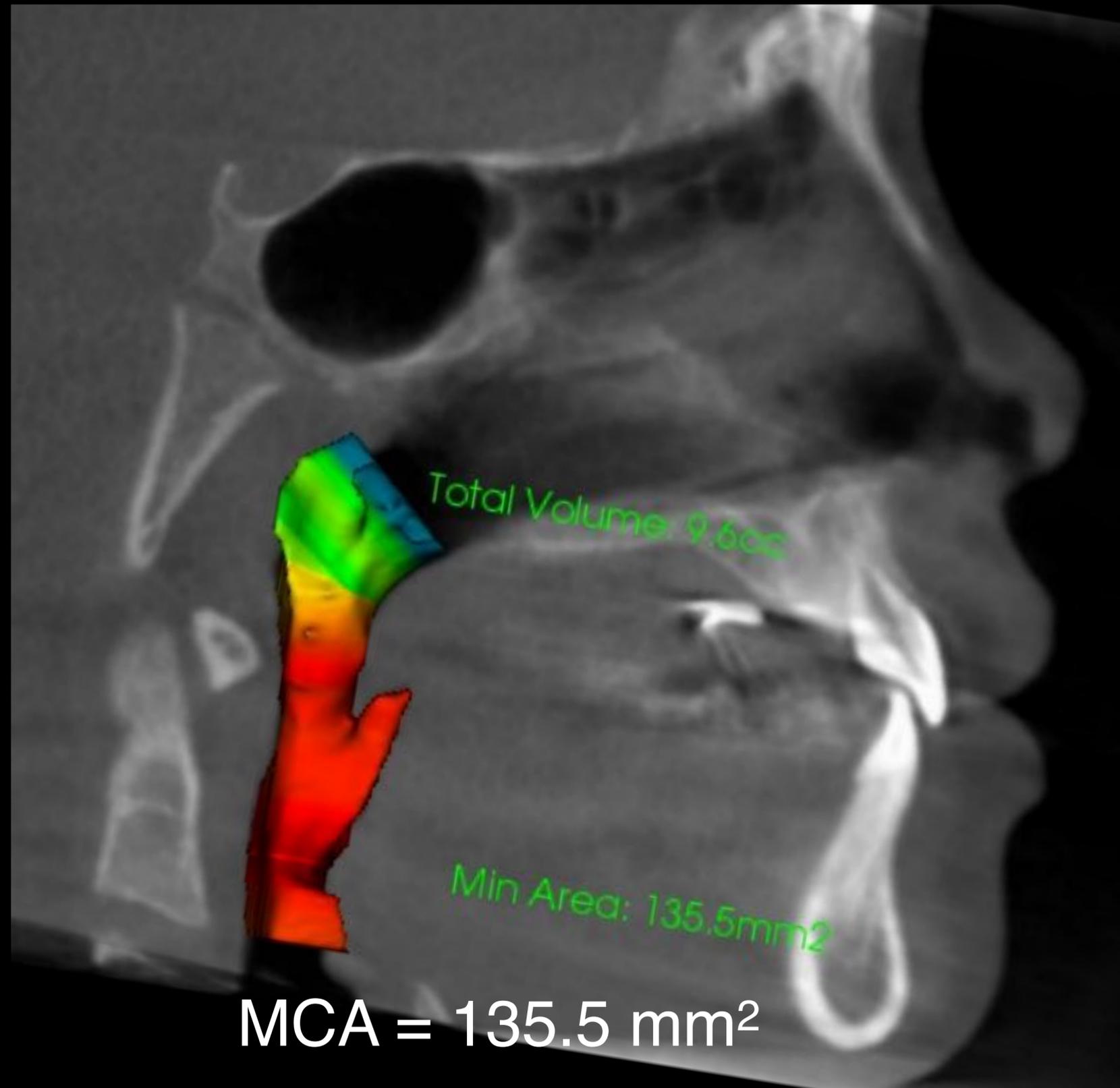
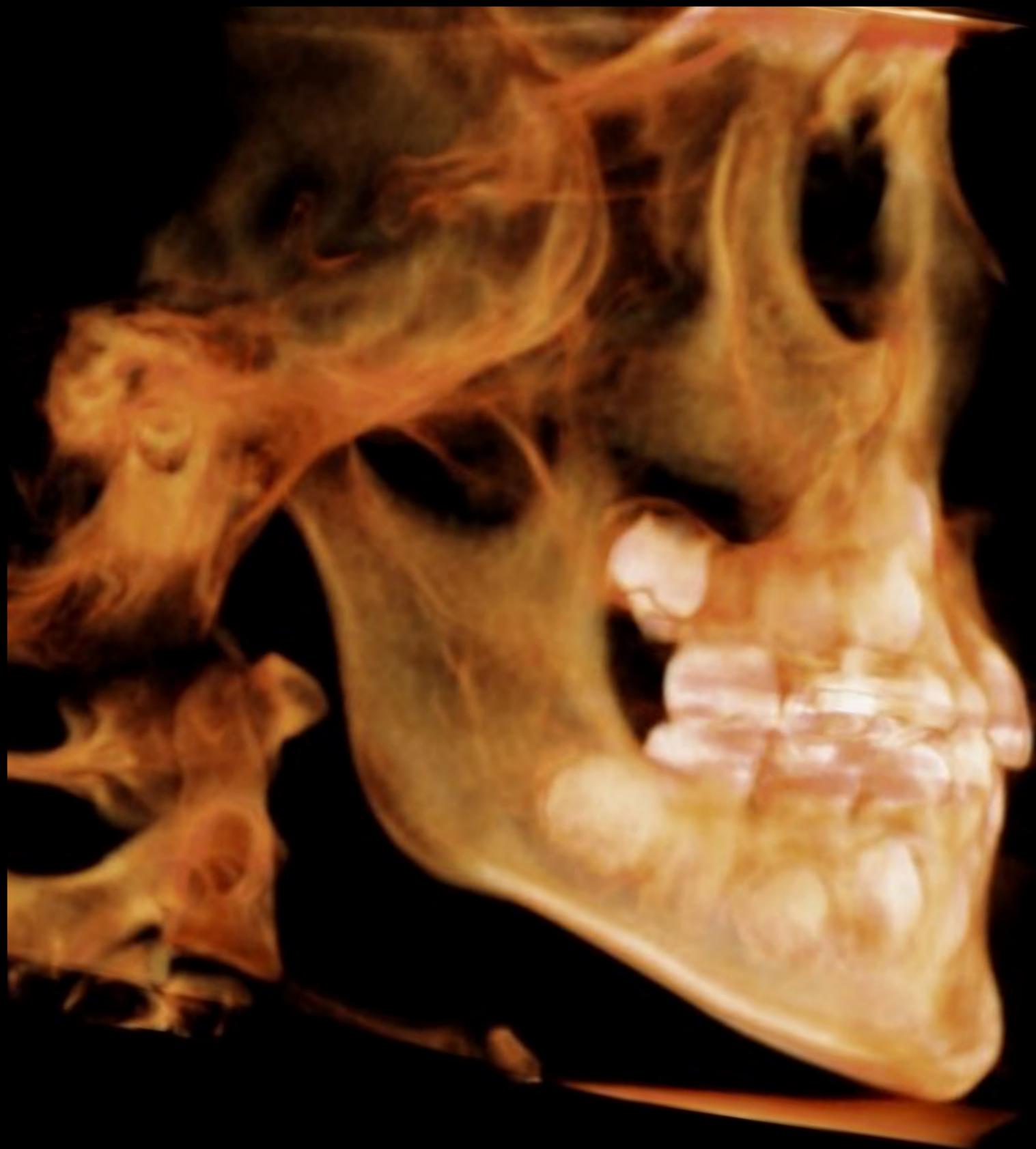
Post-treatment MCA = 304 mm²

Slide courtesy of Dr. J.C. Quintero

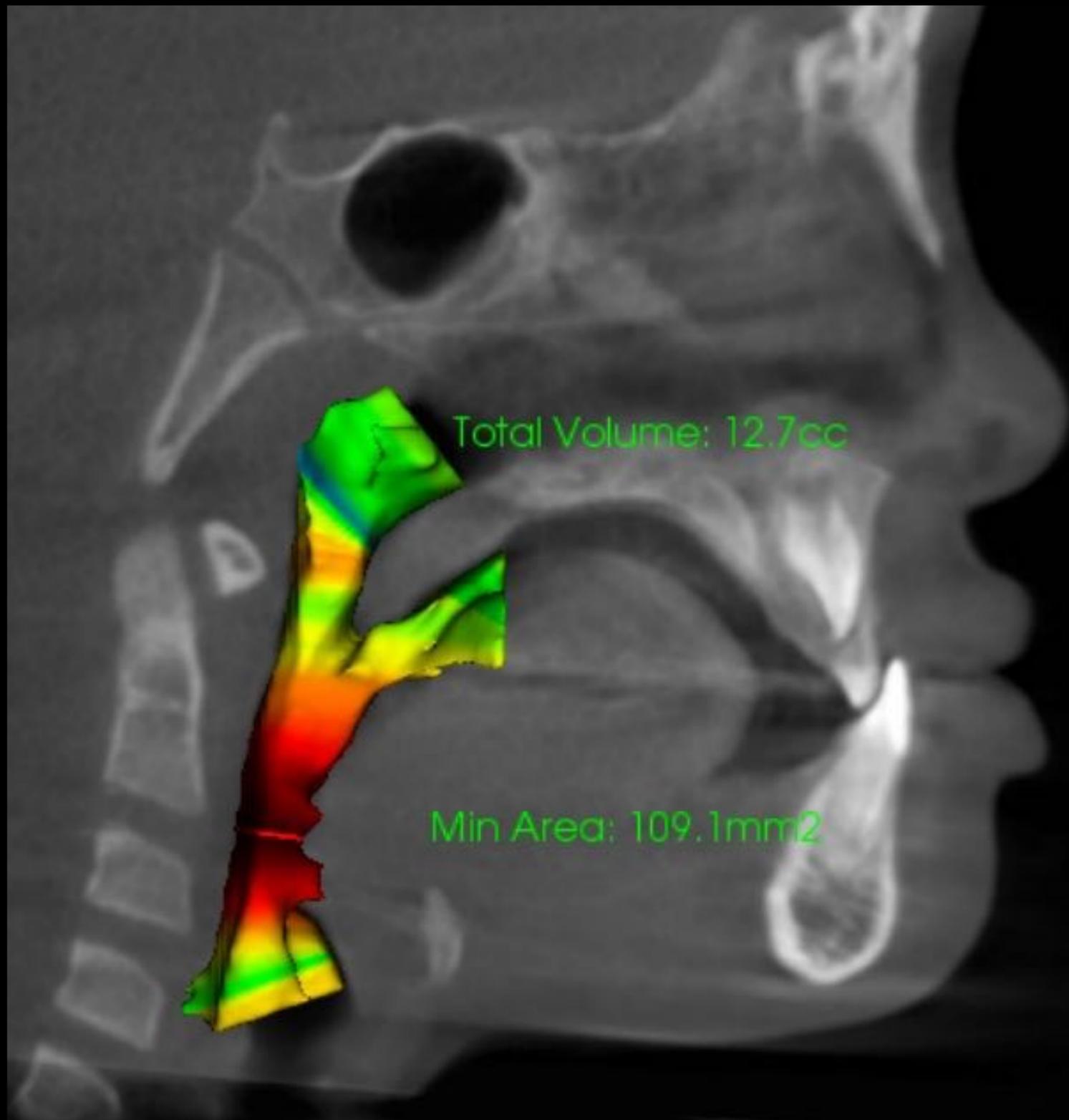




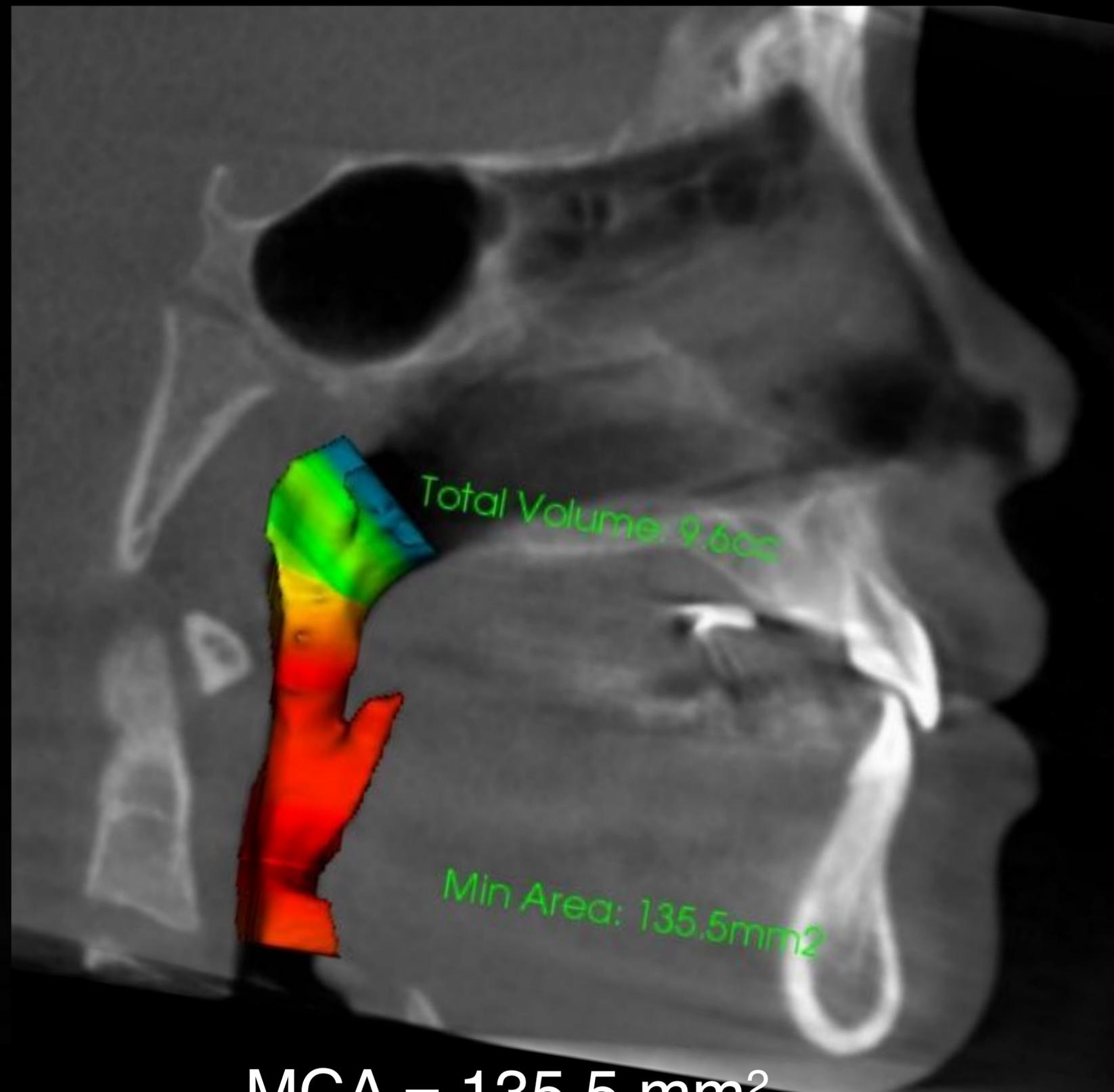




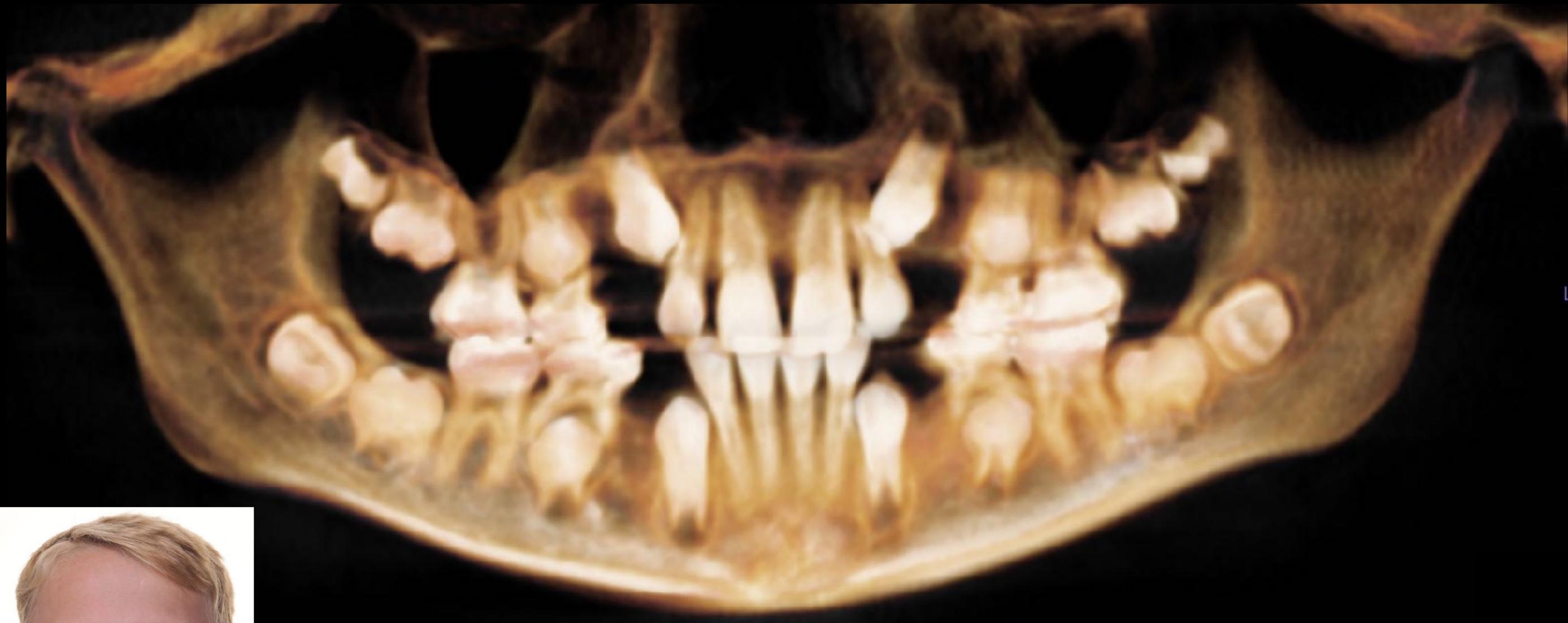
MCA = 135.5 mm²



MCA = 109.1 mm²



MCA = 135.5 mm²



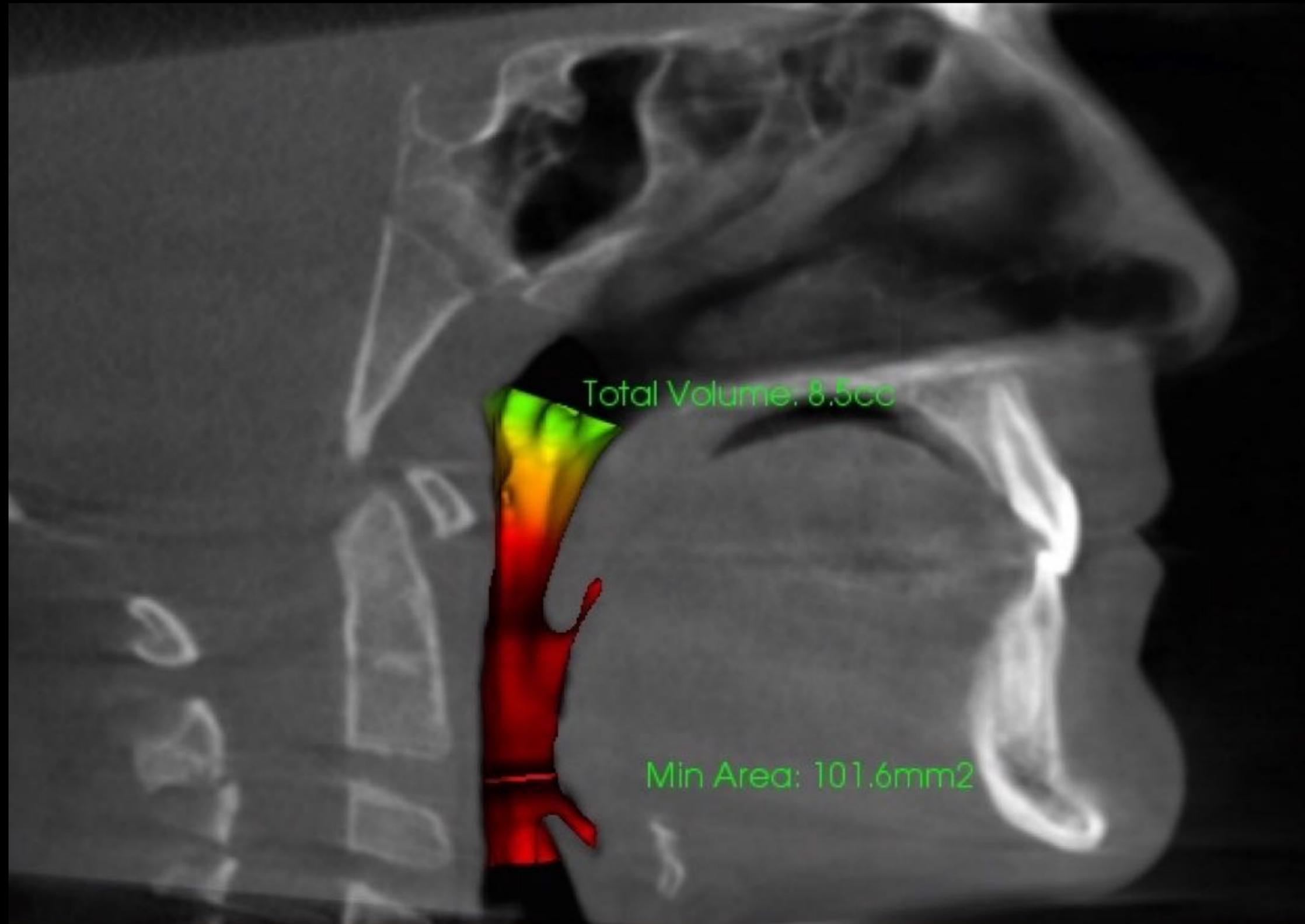
OSA Risk

High Risk = 0-52 mm²

Moderate Risk = 52-110 mm²

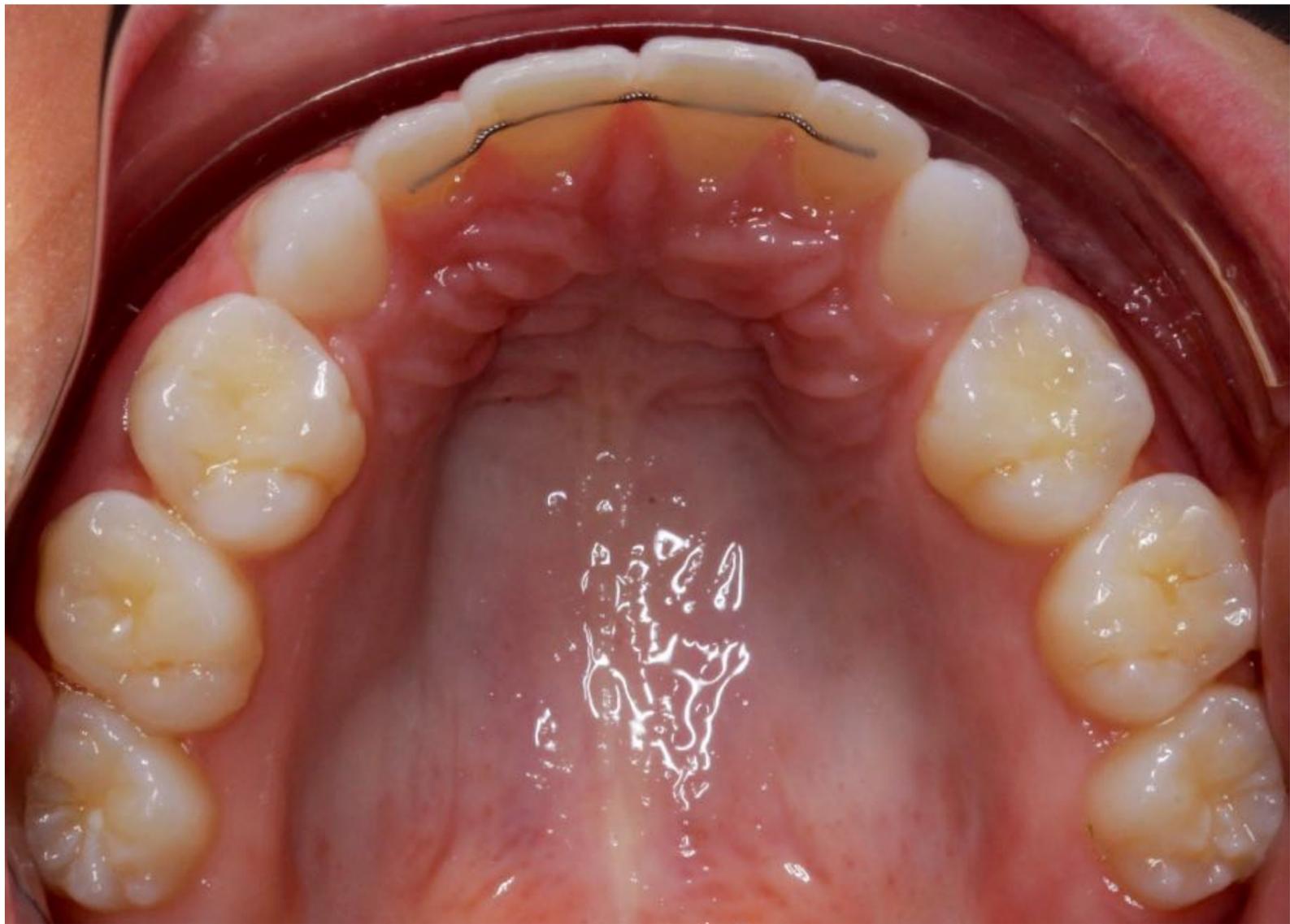
Low Risk > 110 mm²

MCA = 101.6 mm²



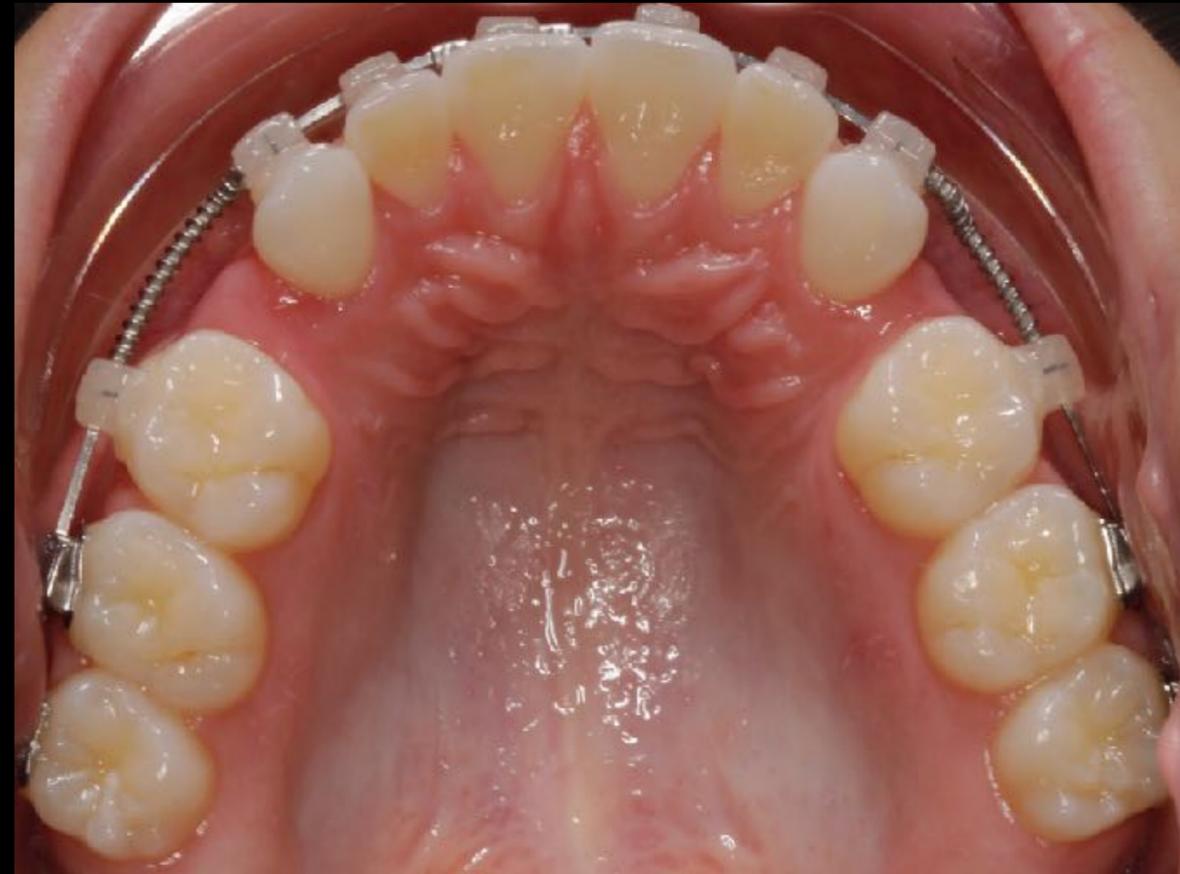
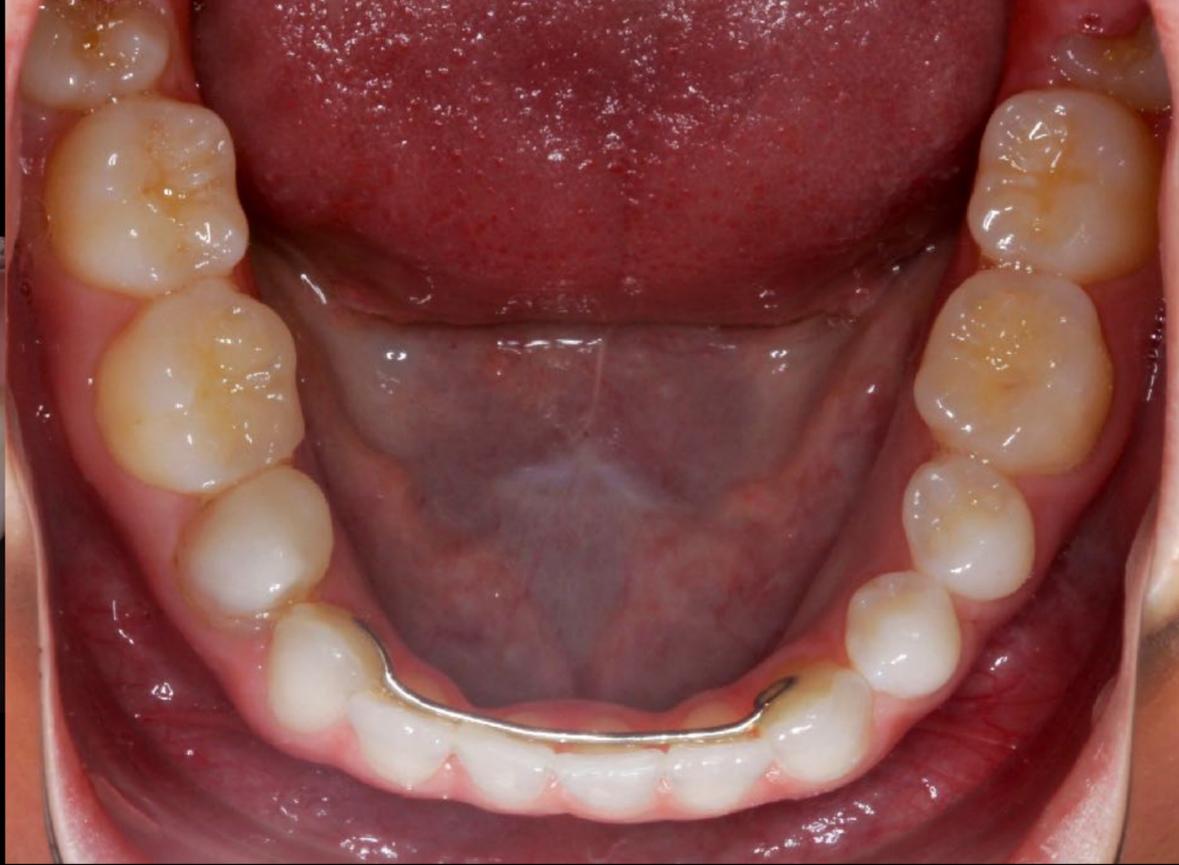
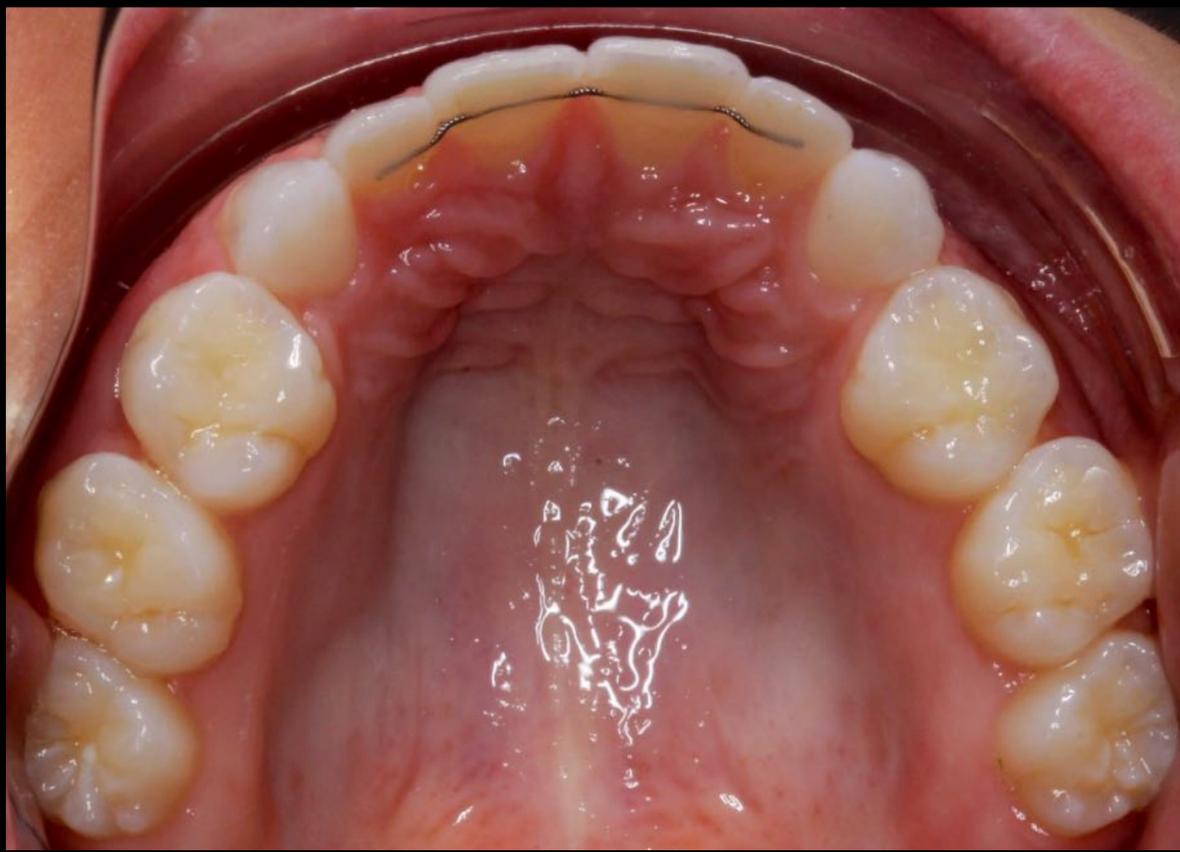






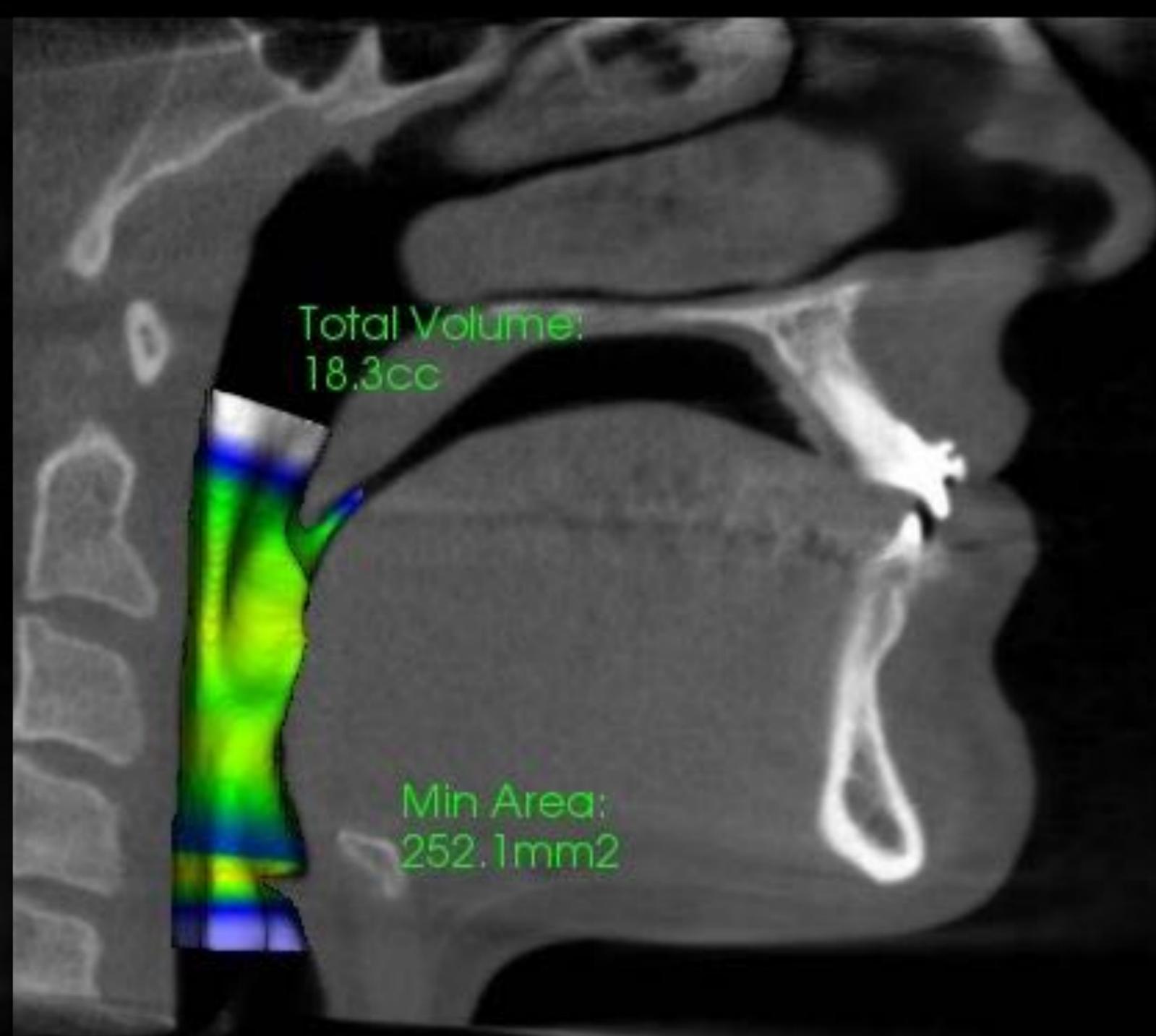
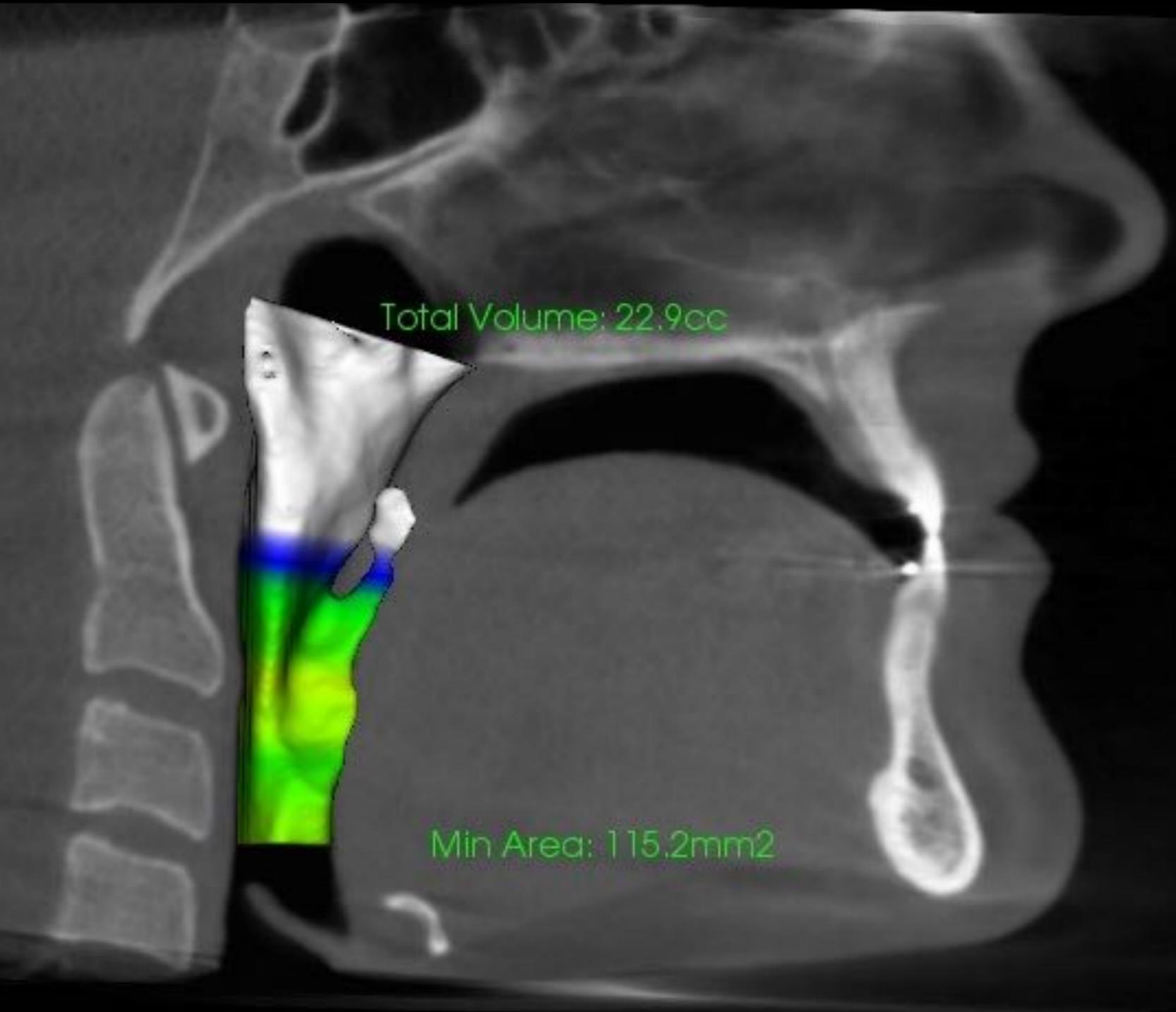






Initial

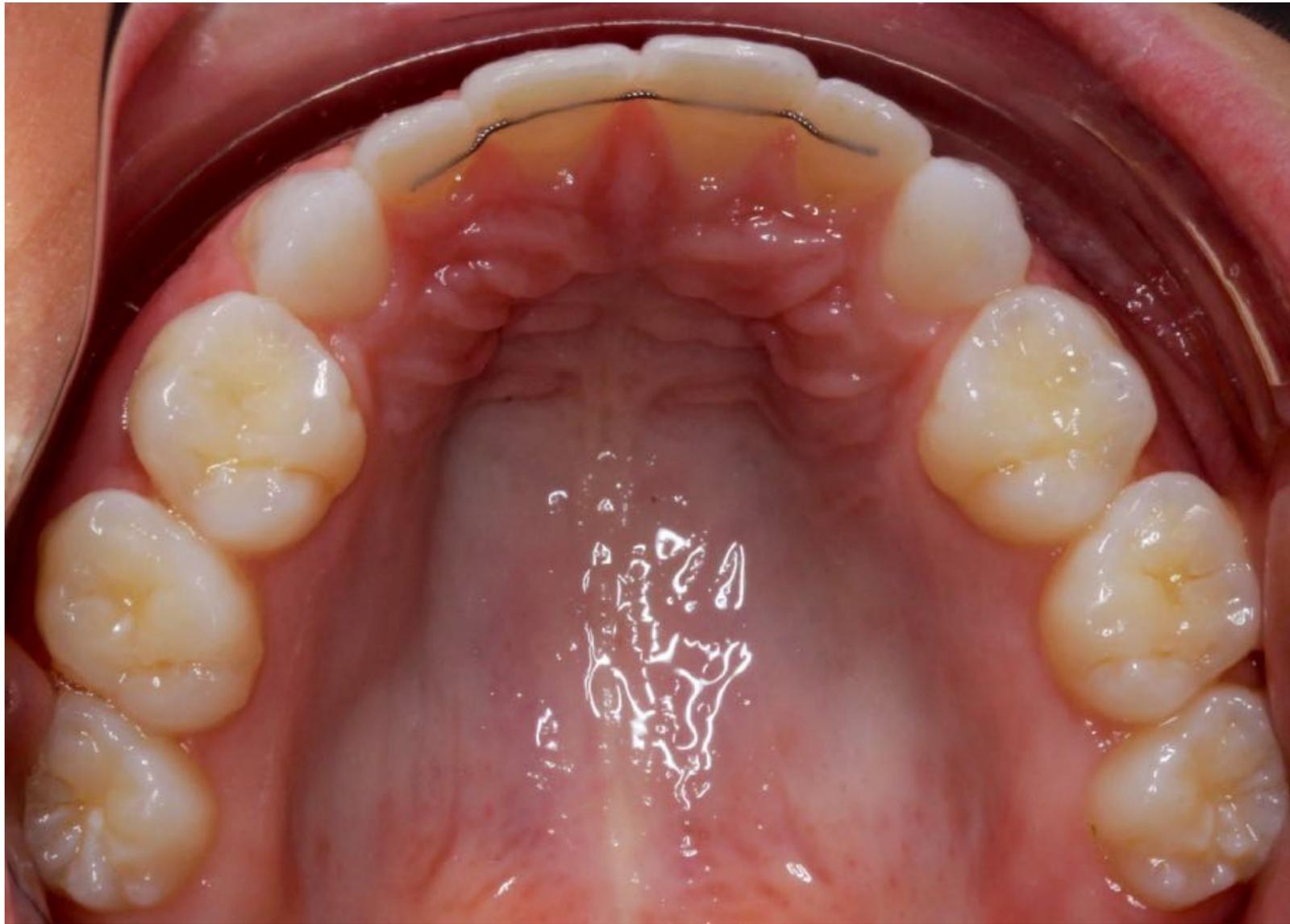
Progress



MCA = 115.2 mm²

MCA = 252.1 mm²







ATOM (Child) Phantom

OSL ID No.	Child Phantom Location (level of OSLD location)
1	Calvarium anterior (2)
2	Calvarium left (2)
3	Calvarium posterior (2)
4	Mid brain (2)
5	Mid brain (3)
6	Pituitary (4)
7	Right orbit (4)
8	Right lens of eye (4-5)
9	Left lens of eye (4-5)
10	Right maxillary sinus (5)
11	Left nasal airway (5)
12	Right parotid (6)
13	Left parotid (6)
14	Left back of neck (6)
15	Right ramus (7)
16	Left ramus (7)
17	Right submandibular gland (7)
18	Left submandibular gland (7)
19	Center sublingual gland (7)
20	Center C spine (8)
21	Thyroid superior-left (8)
22	Thyroid - left (9)
23	Thyroid - right (9)
24	Esophagus (9)

Phantom Levels



Slide courtesy of Dr. Sean Carlson

How many microseiverts
does a child receive with
an 4.8 second scan?

17.5

μSv

The American Association of Physicists in Medicine (AAPM) issued the following position statement in December, 2011: “risks of medical imaging at effective doses below 50,000 μSv for single procedures or 100,000 μSv for multiple procedures over short time periods are too low to be detectable and may be nonexistent.”¹

The ICRP recommends we keep non-occupational exposure levels less than 1,000 $\mu\text{Sv}/\text{pt.}/\text{year}$.²

The The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) report of 2012 states that no discernible effects of exposures below 0.1 Sv (100,000 μSv) appear to exist, which is compatible with known cellular-repair mechanisms.³

1. Dezarn, W. A. *et al.* Recommendations of the American Association of Physicists in Medicine on dosimetry, imaging, and quality assurance procedures. *Medical physics* **38**, 4824–4845 (2011).

2. ICRP, 2007. 2007 Recommendations of the International Commission on Radiological Protection (Users Edition). ICRP Publication 103 (Users Edition). Ann. ICRP 37 (2-4).

3. Bertin, D. M. [2012 report of UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation)]. 1–21 (2012).

All Patients In My Practice
Have A CBCT Scan

If CBCT Scan Reveals Risk

Pediatric Sleep Questionnaire: Sleep-Disordered Breathing Subscale 070129

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JOHN GRAHAM DDS MD

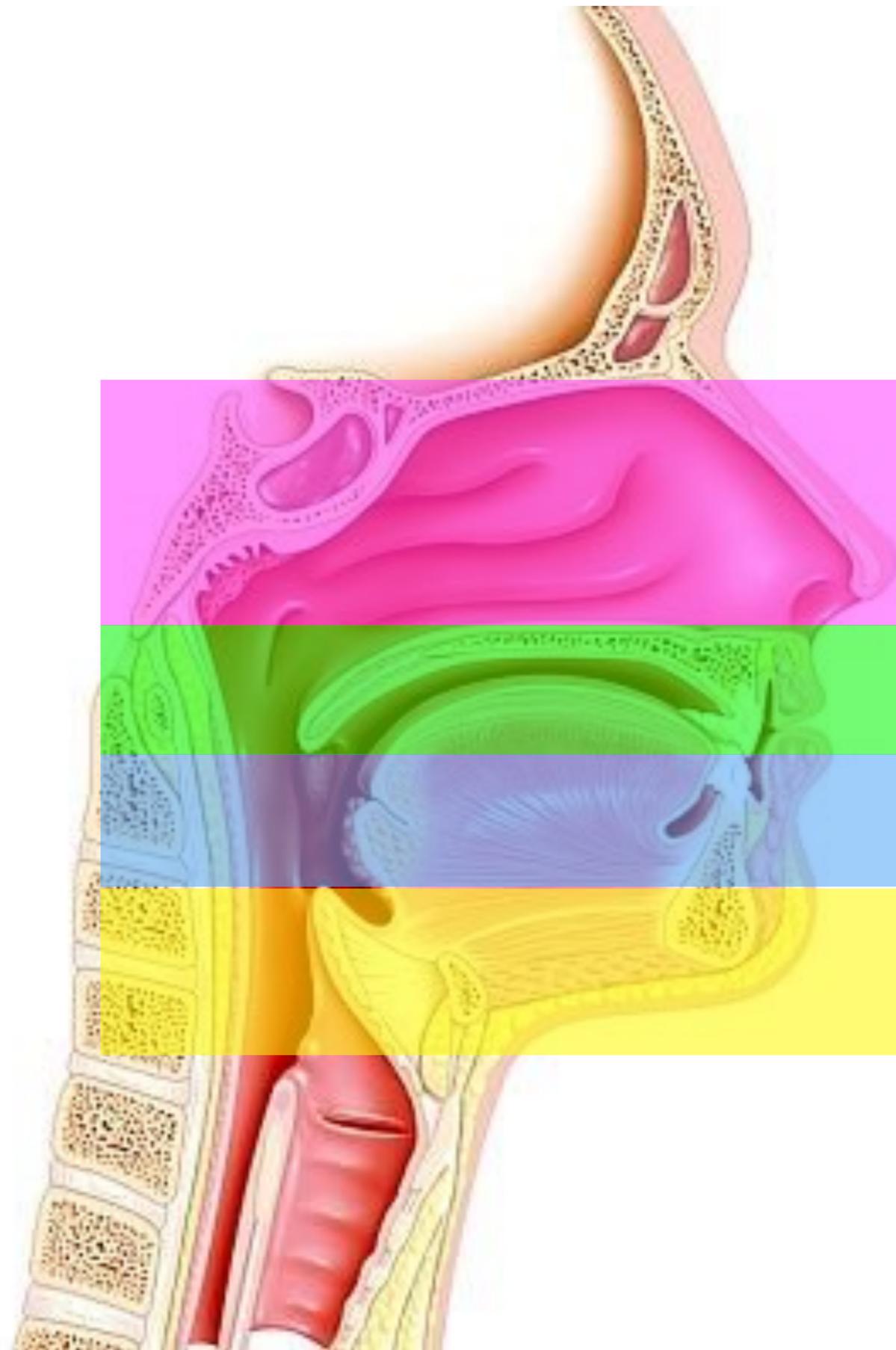
Child's Name: _____ Study ID #: _____
 Person completing form: _____ Date: ____/____/____

Please answer these questions regarding the behavior of your child during sleep and wakefulness. The questions apply to how your child acts in general during the past month. You should circle the correct response. A "Y" means "yes," "N" means "no," and "DK" means "don't know."

1. WHILE SLEEPING, DOES YOUR CHILD:				
Snore more than half the time?	Y	N	DK	A2
Always snore?	Y	N	DK	A3
Snore loudly?	Y	N	DK	A4
Have "heavy" or loud breathing?	Y	N	DK	A5
Have trouble breathing, or struggle to breathe?	Y	N	DK	A6
2. HAVE YOU EVER SEEN YOUR CHILD STOP BREATHING DURING THE NIGHT?				
	Y	N	DK	A7
3. DOES YOUR CHILD:				
Tend to breathe through the mouth during the day?	Y	N	DK	A24
Have a dry mouth on waking up in the morning?	Y	N	DK	A25
Occasionally wet the bed?	Y	N	DK	A32
4. DOES YOUR CHILD:				
Wake up feeling unrefreshed in the morning?	Y	N	DK	B1
Have a problem with sleepiness during the day?	Y	N	DK	B2
5. HAS A TEACHER OR OTHER SUPERVISOR COMMENTED THAT YOUR CHILD APPEARS SLEEPY DURING THE DAY?				
	Y	N	DK	B4
6. IS IT HARD TO WAKE YOUR CHILD UP IN THE MORNING?				
	Y	N	DK	B6
7. DOES YOUR CHILD WAKE UP WITH HEADACHES IN THE MORNING?				
	Y	N	DK	B7
8. DID YOUR CHILD STOP GROWING AT A NORMAL RATE AT ANY TIME SINCE BIRTH?				
	Y	N	DK	B9
9. IS YOUR CHILD OVERWEIGHT?				
	Y	N	DK	B22
10. THIS CHILD OFTEN:				
Does not seem to listen when spoken to directly.	Y	N	DK	C3
Has difficulty organizing tasks and activities.	Y	N	DK	C5
Is easily distracted by extraneous stimuli.	Y	N	DK	C8
Fidgets with hands or feet or squirms in seat.	Y	N	DK	C10
Is "on the go" or often acts as if "driven by a motor".	Y	N	DK	C14
Interrupts or intrudes on others (eg., butts into conversations or games).	Y	N	DK	C18

The 22 items of the SRBD Scale are each answered yes = 1, no = 0, or don't know = missing. The number of symptom-items endorsed positively ("yes") is divided by the number of items answered positively or negatively; the denominator therefore excludes items with missing responses and items answered as don't know. The result is a number, a proportion that ranges from 0.0 to 1.0. Scores > 0.33 are considered positive and suggestive of high risk for a pediatric sleep-related breathing disorder. This threshold is based on a validity study that suggested optimal sensitivity and specificity at the 0.33 cut-off.¹

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Nasopharynx

Velopharynx

Oropharynx

Hypopharynx

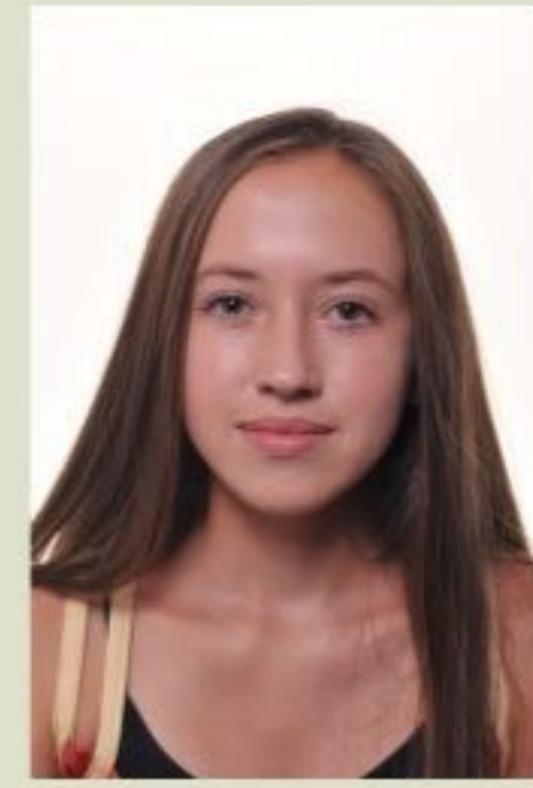
Initial (16, 8/11/15)

Most Recent Set

08/11/15



Browser... Most Recent



No Tx Note for Today

Approve Treatment Note

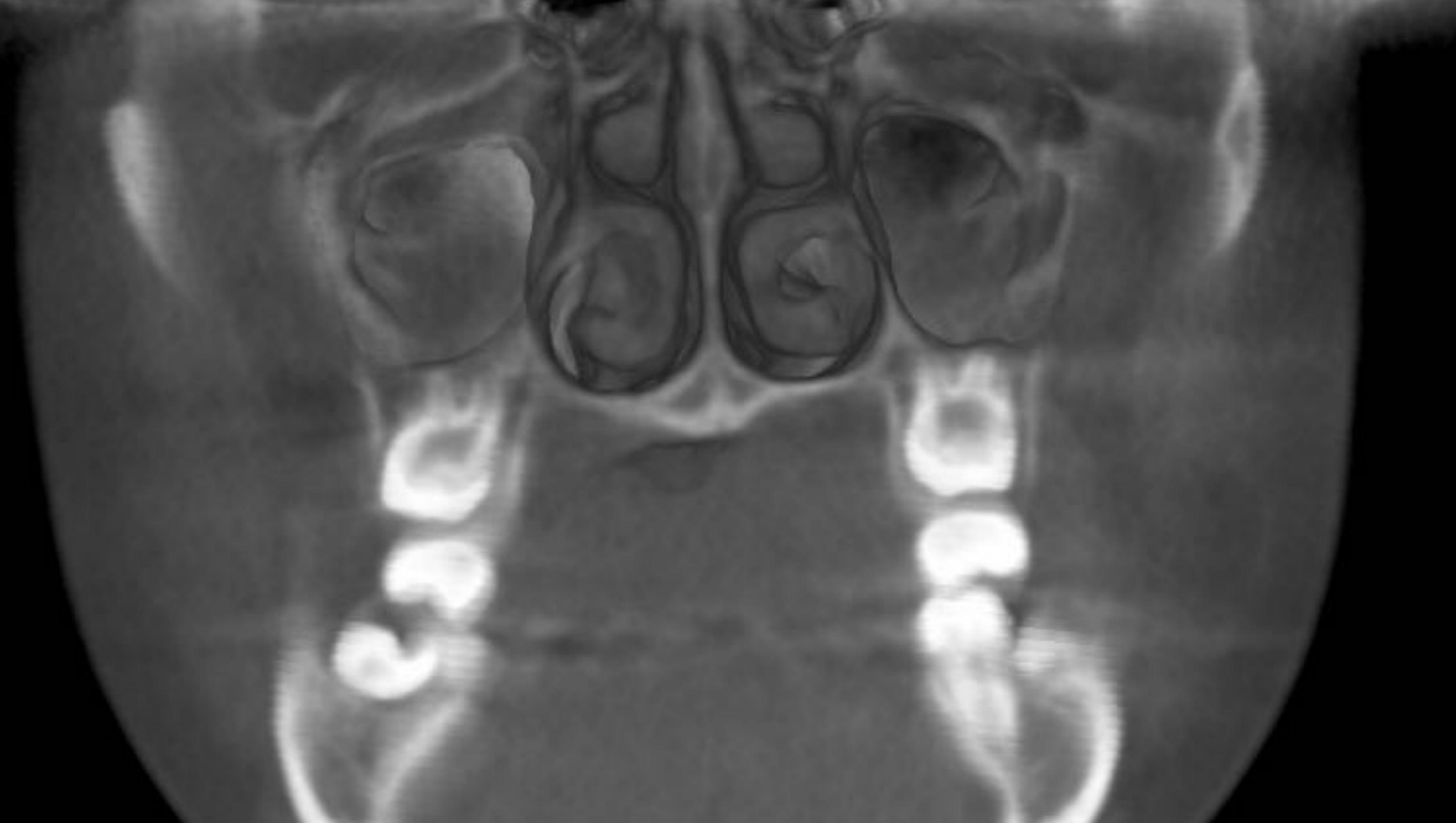
Approval	Date	Compliance	Appliances	Elastics	AWs	Status	Notes	Next Appt	Next Appt Notes
	08/11/15 JG/CG					Active Tr... Started	CT SCAN, PHOTOS, EXAM. HAD BRACES FOR 3 YRS WITH DR [REDACTED] AT [REDACTED] BITE IS STILL OFF, TEETH ARE FLARED. SHE WAS NEVER GIVEN ANY RETAINERS, ONLY PUT FIXED RETAINERS ON CHLOE.		

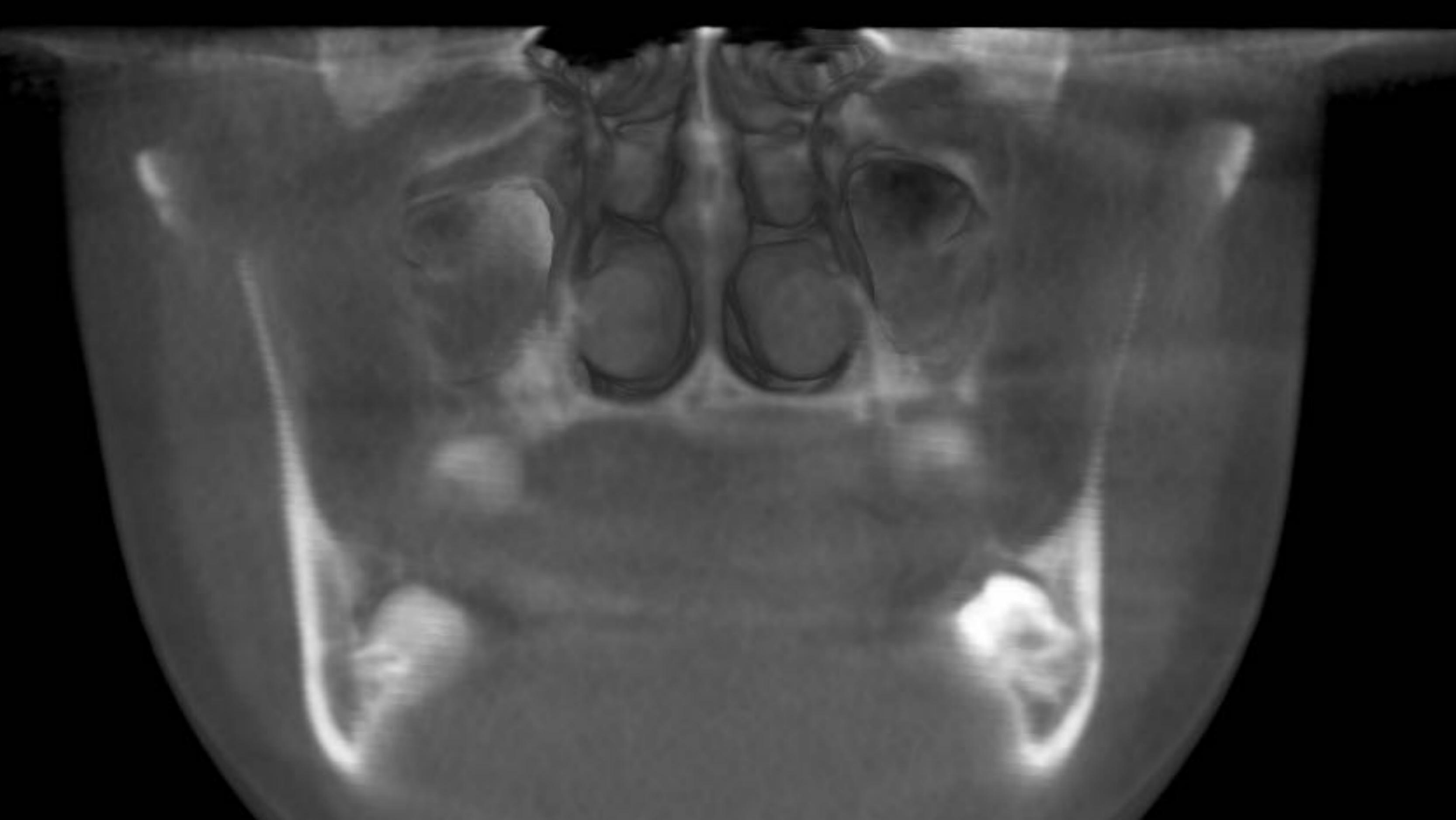










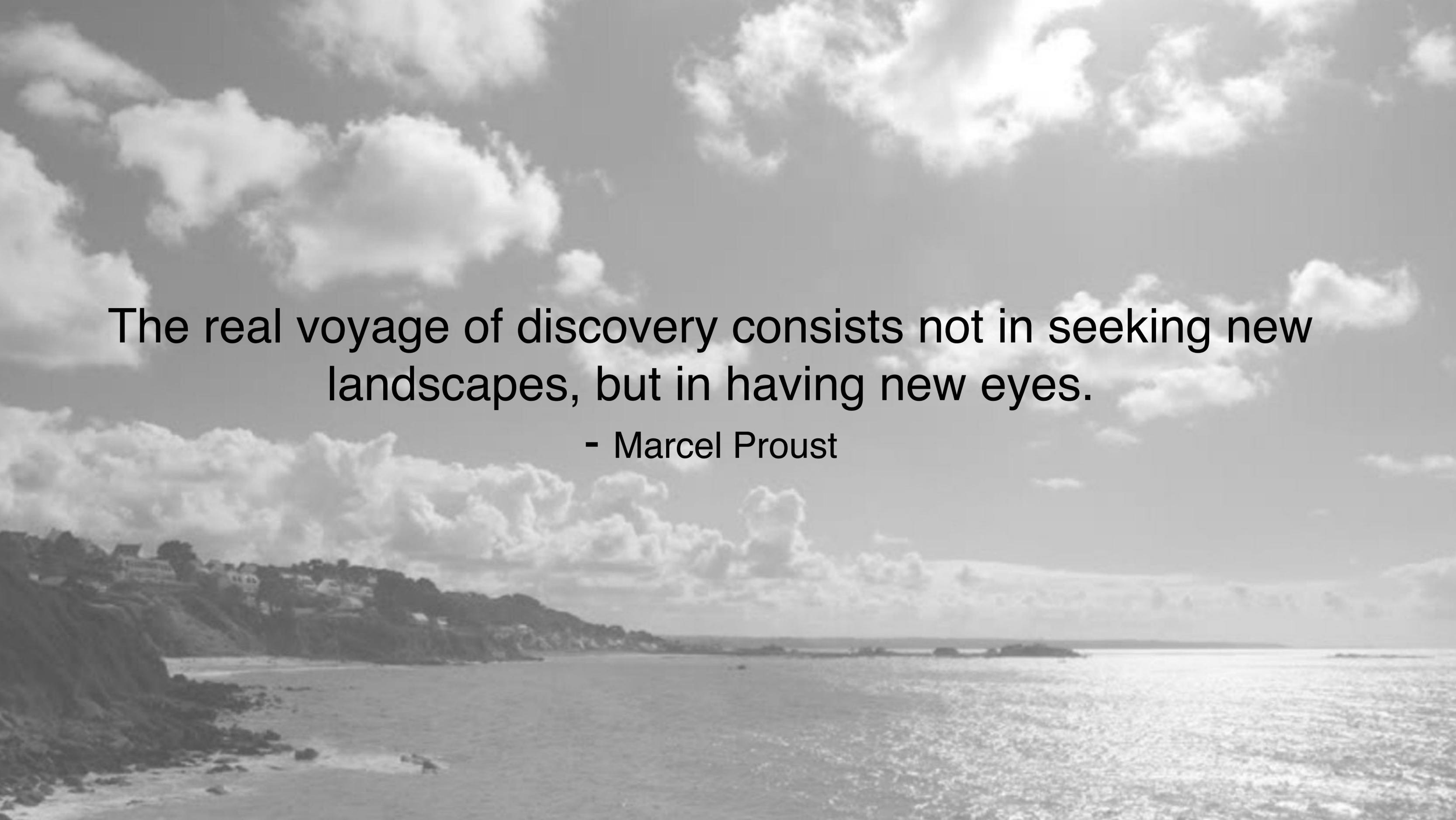


My daughter Chloe has TRULY never been able to breathe through her nose since birth. At an early age we noticed how she would breathe through her mouth while awake, as well as while she slept. She never learned to blow her nose, and when eating she would have to stop chewing sometimes to take a breath. She was also susceptible to strep because of her mouth breathing. If she was sick with a cold, she had absolutely no chance. As a small child she would look up at me with tears and say “All I want is a new nose.”

All of this lead to many doctor visits: ENT's, Allergists, cameras place up her nose, which lead to different diagnoses and a plethora of medications. She learned to adjust her lifestyle based on a Severe Allergy diagnosis. Her daily regimen was saline nasal cleanse x 2/day, Nasacort, Claritin, Sudafed, an occasional Benadryl, not to mention the Afrin as needed. She also slept sitting up.

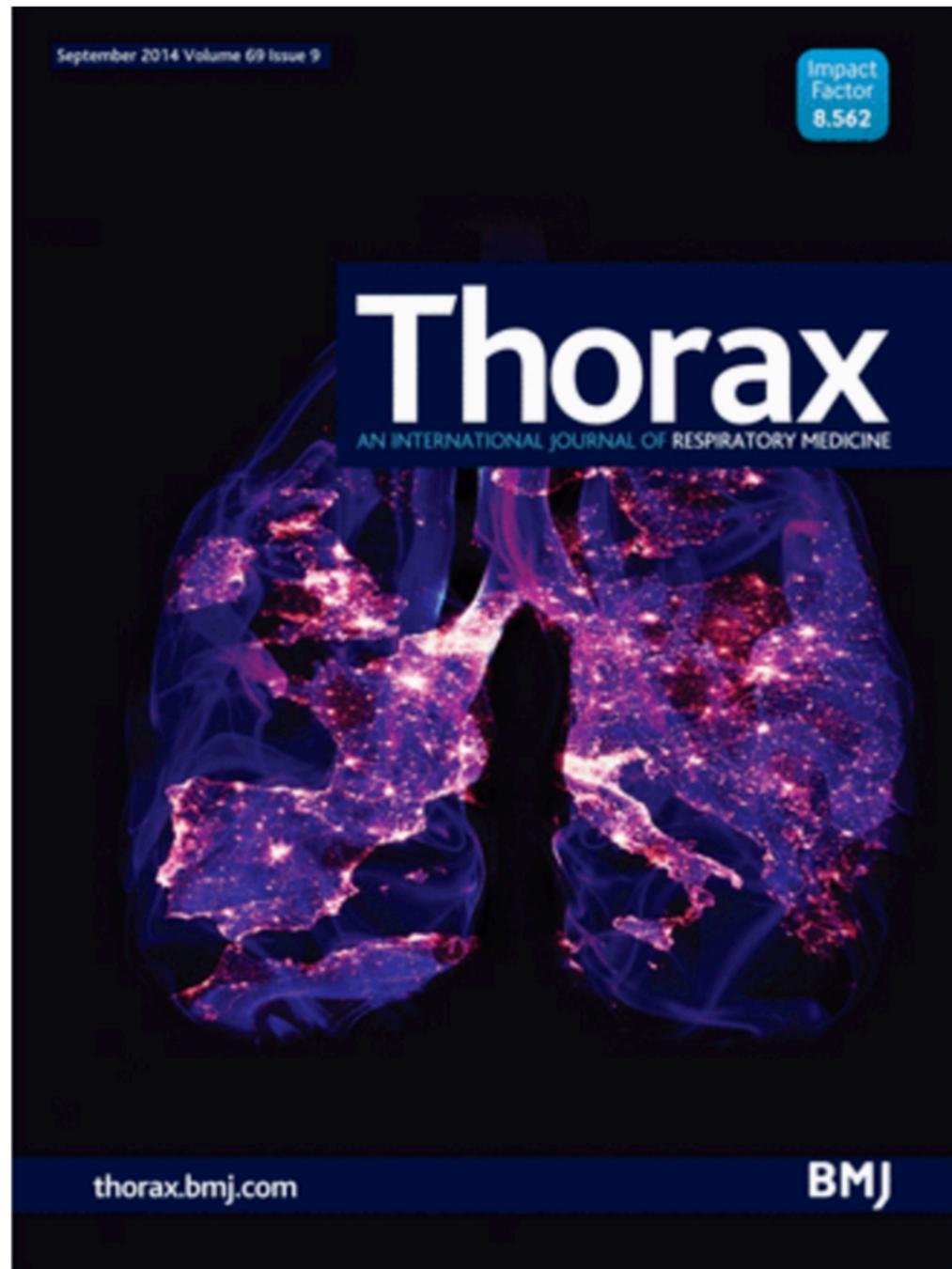
We are grateful to you Dr. Graham for taking time to care and take interest in Chloe. A week after surgery and the removal of splints, Chloe had the results she always dreamed of. She sleeps on one pillow, breathes with her mouth closed, blew her nose for the first time, no dry lips or dry mouth, and she is 100% medication-free. We think of putting all that medication in her body and all the money spent and it's very unsettling.

This has been life changing for her. Thanks Dr. Graham, for giving Chloe her “new nose”!



The real voyage of discovery consists not in seeking new
landscapes, but in having new eyes.

- Marcel Proust



The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea

Andrew S L Chan,^{1,2,3} Kate Sutherland,^{1,2} Richard J Schwab,⁴ Biao Zeng,^{1,2,3} Peter Petocz,⁵ Richard W W Lee,^{1,2,3} M Ali Darendeliler,⁶ Peter A Cistulli^{1,2,3}

ABSTRACT

Background The mechanisms by which mandibular advancement splints (MAS) improve obstructive sleep apnoea (OSA) are not well understood. This study aimed to evaluate the mechanism of action of MAS by assessing their effect on upper airway structure in patients with OSA.

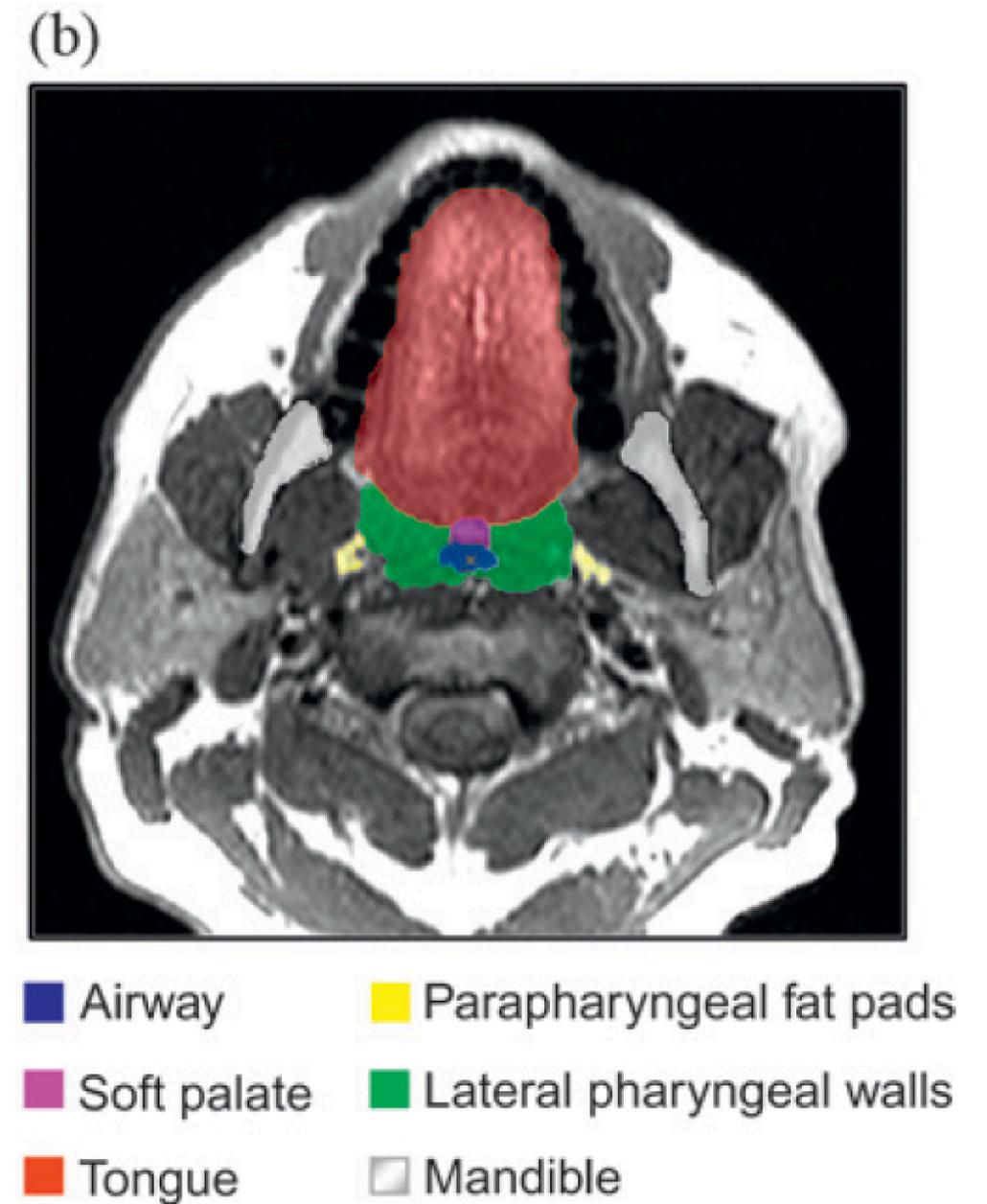
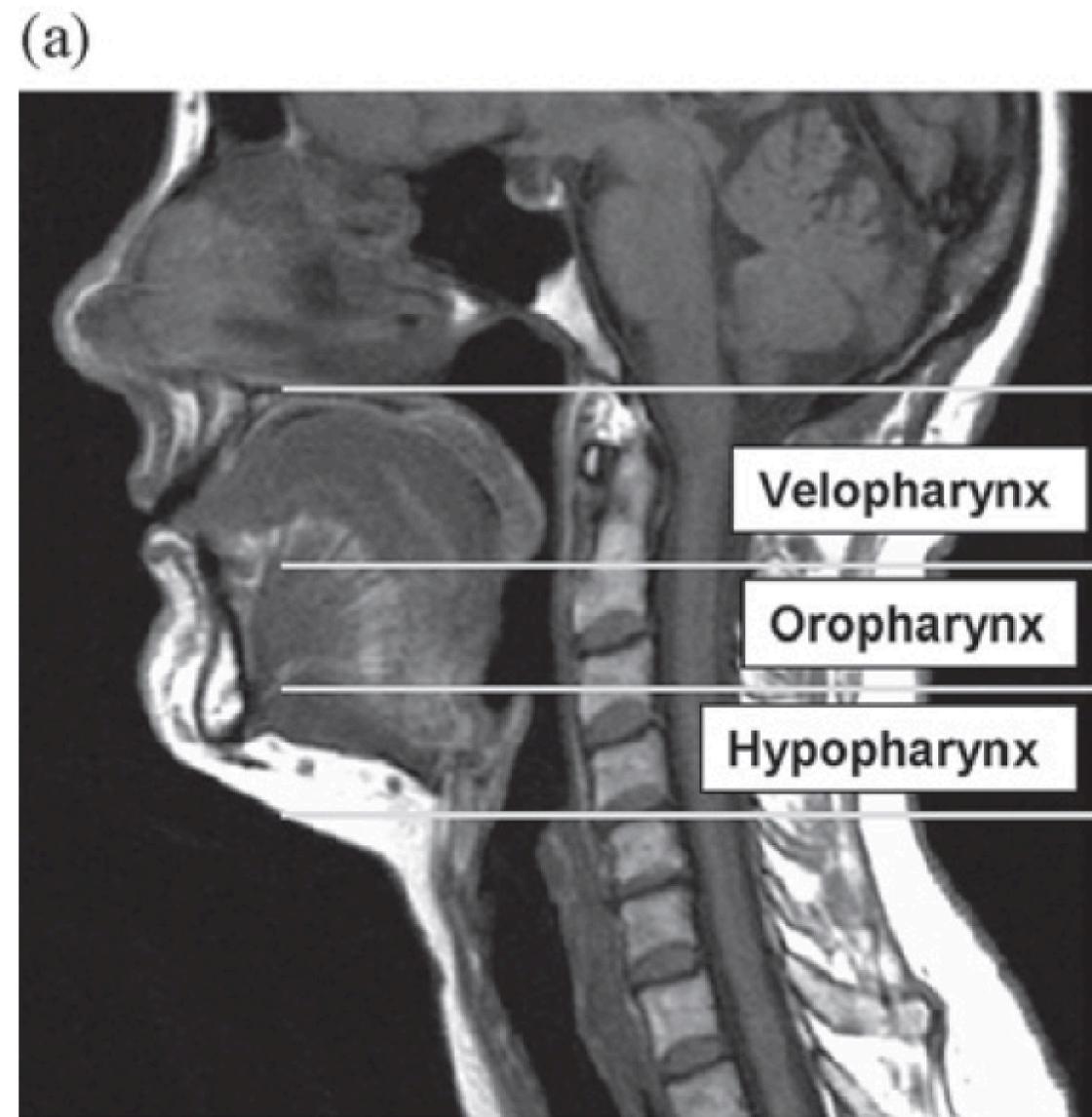
Methods Patients were recruited from a sleep disorders clinic for treatment with a custom-made MAS. MRI of the upper airway was performed during wakefulness in the supine position, with and without the MAS.

Results Sixty-nine patients with OSA were recruited. Treatment with the MAS reduced the apnoea–hypopnoea index (AHI) from 27.0 ± 14.7 events/h to 12.2 ± 12.5 events/h ($p < 0.001$). There was an increase in the total airway volume with mandibular advancement (16.5 ± 0.7 cm³ vs 18.1 ± 0.8 cm³; $p < 0.01$) that occurred predominantly because of an increase in the volume of the velopharynx (5.7 ± 0.3 cm³ vs 6.5 ± 0.3 cm³; $p < 0.001$). This increase in airway calibre was associated with an increase in the lower anterior facial height (6.8 ± 0.1 cm vs 7.5 ± 0.1 cm; $p < 0.001$), reduction in the distance between the hyoid and posterior nasal spine (7.4 ± 0.1 cm vs 7.2 ± 0.1 cm; $p < 0.001$), lateral displacement of the parapharyngeal fat

preventing collapse of the upper airway during sleep.⁴ However, the mechanisms by which MAS improve OSA are not well understood. Limited studies have identified an effect of mandibular advancement on aspects of the structure and function of the upper airway.^{9–16} Importantly, these predominantly used cephalometric x-rays which are limited by their two-dimensional nature. However, soft tissue volumes and movements, and the interaction between upper airway structural parameters and treatment response have never been systematically studied in patients using an oral appliance. A better understanding of the biomechanical mechanisms that mediate the efficacy of MAS may have important clinical implications, including the development of more efficacious appliances, and may improve the selection of patients for this treatment modality.

MRI is a powerful, non-invasive research tool and is probably one of the best methods for assessing the three-dimensional structure of the upper airway lumen and the surrounding soft tissue structures.¹⁷ Therefore, this study aimed to evaluate the mechanism of action of MAS in patients with OSA by assessing their effect on

Figure 2 (A) Segments of the upper airway on mid-sagittal MRI: velopharynx (from the hard palate to the tip of the uvula), oropharynx (from the tip of the uvula to the tip of the epiglottis) and hypopharynx (from the tip of the epiglottis to the level of the vocal cords). (B) Segmentation of upper airway structures on axial MRI: airway lumen, parapharyngeal fat pads, soft palate, tongue (genioglossus) and lateral pharyngeal walls.



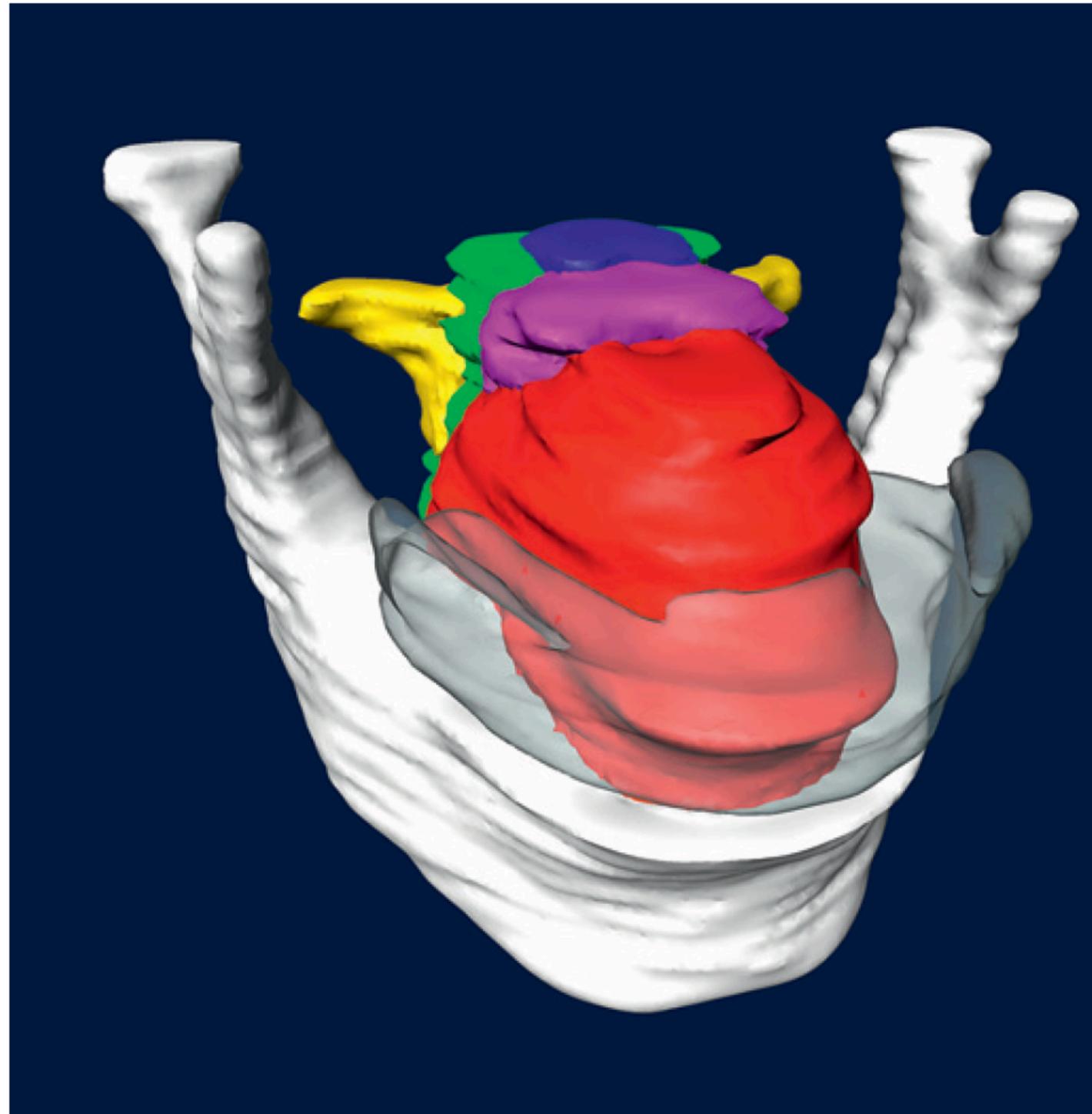
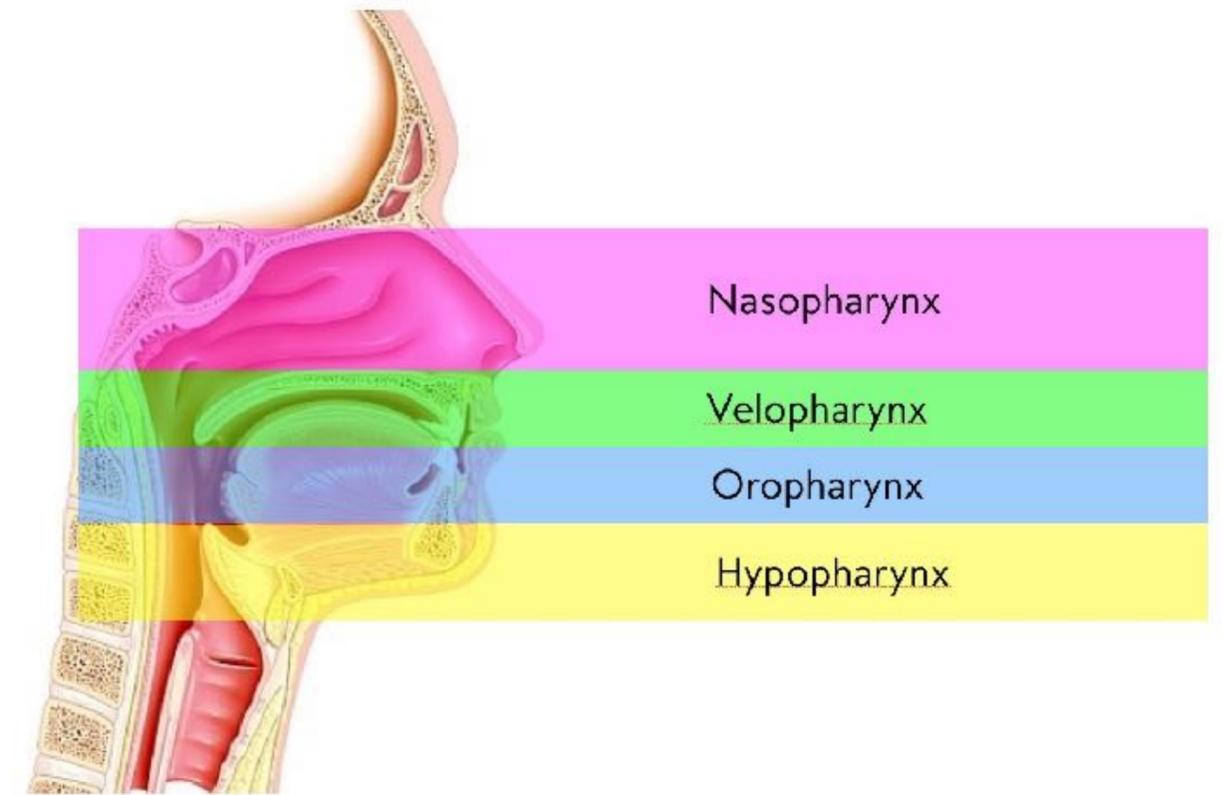
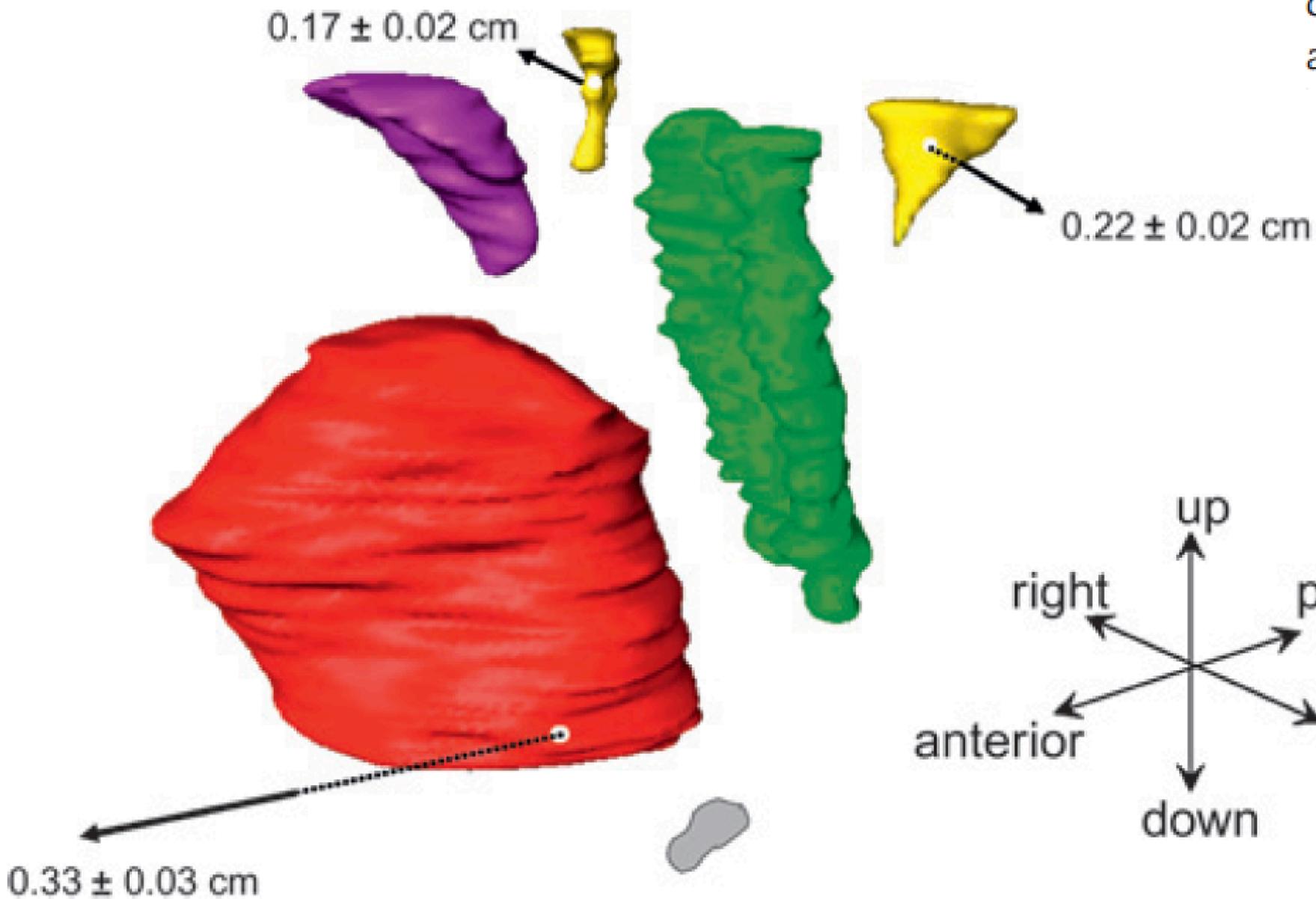


Figure 4 Volumetric reconstruction of the upper airway. The structures shown are: airway lumen (blue), soft palate (purple), tongue (red), parapharyngeal fat pads (yellow), lateral pharyngeal walls (green) and mandible (white). The MAS is shown in grey.

during sleep.⁴ A key finding of this study is that mandibular advancement improves the calibre of the upper airway, but this occurs predominantly due to an increase in the volume of the velopharynx and is mediated by an increase in its lateral dimensions. These airway effects are associated with a number of bony and soft tissue changes, including an increase in lower anterior facial height, raised position of the hyoid, lateral displacement of the parapharyngeal fat pads away from the airway and anterior positioning of the base of tongue muscles.



Does the orthodontic specialty get
it?



Early orthodontic treatment for Class II malocclusion reduces the chance of incisal trauma: Results of a Cochrane systematic review

Badri Thiruvengkatachari,^a Jayne Harrison,^b Helen Worthington,^c and Kevin O'Brien^d

Manchester and Liverpool, United Kingdom

In this article, we summarize the most clinically relevant findings of our recently updated Cochrane systematic review into the treatment of Class II Division 1 malocclusion. **Methods:** A systematic review of the databases was performed to identify all randomized controlled trials evaluating early treatment with functional appliances to correct Class II Division 1 malocclusion. **Results:** Three early treatment studies with data from 353 participants were included in this review. The results showed no significant difference for any outcomes, except new incidence of incisor trauma, which was significantly less for the early treatment group. The risk ratio analysis for new incisor trauma showed that providing early treatment reduced the risk of trauma by 33% and 41% in the functional and headgear groups, respectively. However, when the numbers needed to treat were calculated, early treatment with functional appliances prevents 1 incidence of incisal trauma for every 10 patients (95% CI, 5-174), and headgear treatment prevents 1 incidence of incisal trauma for every 6 patients (95% CI, 3-23). **Conclusions:** Orthodontic treatment for young children, followed by a later phase of treatment when the child is in early adolescence, appears to reduce the incidence of new incisal trauma significantly compared with treatment that is provided in 1 phase when the child is in early adolescence. However, these data should be interpreted with caution because of the high degree of uncertainty. There are no other advantages in providing 2-phase treatment compared with 1 phase in early adolescence. (Am J Orthod Dentofacial Orthop 2015;148:47-59)

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**Which specialty is best
poised to discover, treat
and follow-up with sleep
disordered breathing in
children?**

Sleep Physician



Sleep Testing/Screening when referred by pediatrician

Cannot prescribe, insert or adjust oral appliances

Referral for adenotonsillectomy

Nasal corticosteroids

Sleep Dentist



Referral to sleep specialist

Referral to orthodontist

Orthodontist



CBCT airway evaluation on every patient

Sleep testing/screening

Referral for adenotonsillectomy

Mandibular repositioning

Rapid Maxillary Expansion

Protraction facemask

Orthodontic arch development

iCAT airway re-evaluation

Continued guided airway development into adulthood

Covered by either medical insurance or financed as a phase I

Will



Utah Sleep & Pulmonary Specialists

Utah Sleep & Pulmonary Specialists

Pediatric & Adult Pulmonary & Sleep Medicine

Gregory Dupont, MD
Adult Sleep Specialist
Board Certification:
Pulmonary Medicine
Internal Medicine
Diplomate,
American Board of Sleep Medicine

Kathleen D. Pfeffer, MD
Pediatric Sleep Specialist
Board Certification:
Pulmonary Medicine
Pediatrics
Diplomate,
American Board of Sleep Medicine

Address
9103 S. 1300 W.
Suite 103
West Jordan, UT 84088
801 432-8690
801 432-8681 FAX
UTSleep.com

POLYSOMNOGRAPHIC REPORT

GRAHAM, WILL
DOB: 9-12-02
DOS: 9-20-13

REFERRING PHYSICIAN:

Rachel Baar, M.D.
IHC Memorial Clinic
2000 S. 900 E.
SLC, UT 84105

BRIEF HISTORY: Will is an 11 year old young history of difficulties falling to sleep and maintaining if he is given melatonin, but he still wakes. Parents room and note that he wakes typically between 2 drink of water, goes to his bathroom, but occasionally He states that he can sleep better on the couch sleeping on the floor of his room when more completely denies any symptoms of restless crawlies, and numbness. Paternal grandfather has myasthenia gravis. Maternal grandmother has insomnia. A 3 year old sister is scheduled for

Will gets very frustrated that he cannot sleep very restless, constantly moving. He does not seem to be having difficulty breathing. Tony is having difficulty breathing. Tony's tonsils are circumscripted tonsils, uvula was slightly enlarged. Other findings. Sleep study is performed to determine if a disorder which might contribute to the disorder maintaining sleep.

METHOD: The patient was monitored with conventional polysomnography, including oral/nasal airflow, single channel plethysmography and video recording.

RESULTS: Overall this was a good study. He fell asleep at 10:32 p.m. He fell asleep until 5:06 a.m., or 375 minutes of sleep with an efficiency of 78.1%. He had 2 awakenings when he should have been sleeping. He was horribly restless during stage 3-4 sleep, but around 2:30 a.m. he fell back into stage 2 sleep he transitioned into stage 3-4 sleep again until 3:30 a.m. He had 1 REM cycle. Normal is 20-25%.

Utah Sleep & Pulmonary Specialists

Pediatric & Adult Pulmonary & Sleep Medicine

Gregory Dupont, MD
Adult Sleep Specialist
Board Certification:
Pulmonary Medicine
Internal Medicine
Diplomate,
American Board of Sleep Medicine

Kathleen D. Pfeffer, MD
Pediatric Sleep Specialist
Board Certification:
Pulmonary Medicine
Pediatrics
Diplomate,
American Board of Sleep Medicine

Address
9103 S. 1300 W.
Suite 103
West Jordan, UT 84088
801 432-8690
801 432-8681 FAX
UTSleep.com

PAGE 2 GRAHAM
DOS: 9-20-13

37.5% stage 3+4; and 16.1% REM. REM sleep is low, not surprisingly, given the 1 REM cycle. Normal is 20-25%.

He slept primarily on his back, but associated with transitions to light stage or awakenings he moved back and forth onto his left side.

There are 68 arousals or 13.2 an hour, which is moderately elevated. Of the 11.3 an hour were respiratory event related, also moderately elevated; 7.1 an hour were spontaneous, which is normal.

There were 20 periodic limb movements or 3.9 an hour, which is normal or 0.6 an hour were associated with arousals. However, associated with transitions into stage 1 sleep and light sleep, significant restless leg type activity was noted. In fact 236 leg movements were noted during light sleep or wakefulness and restless legs.

Breathing wise, oxygen saturations asleep on room air average was 93%. There were no obstructive apneic events, there were breathing events occurring primarily during transitions into REM sleep. These were scattered throughout the night; 2 occurred in REM sleep on his back, 2 on his left side. Overall obstructive apnea/hypopnea index (AHI) number of obstructive breathing events per hour, is 1.2, which is normal.

There were 10 central pauses also scattered throughout the night occur during transitions between deep sleep and light sleep. The longest was 14.8 seconds and lasted as long as 14.8 seconds. 3 events occurred on the left side. No central apnea per se, no periodic breathing was noted. The AHI is 1.9. Overall apnea/hypopnea index (AHI) is 3.1, which is normal.

There were 48 RERAs or respiratory event related arousals that cannot be scored as apneas or hypopneas. These typically suggest some degree of upper airway resistance during periods of light stage 1 sleep, and transitions into REM sleep.

Clinical information, periods of increased effort to breathe were noted throughout the night, although no snoring was noted and turning were noted throughout the night.

IMPRESSIONS/RECOMMENDATIONS: The patient does not need to move, these results suggest a disorder consistent with periodic limb movement disorder and disordered breathing.

It should be noted that his long REM sleep was not seen. Both REM sleep and being in the REM sleep and breathing, and this was not seen.

Utah Sleep & Pulmonary Specialists

Pediatric & Adult Pulmonary & Sleep Medicine

Gregory Dupont, MD
Adult Sleep Specialist
Board Certification:
Pulmonary Medicine
Internal Medicine
Diplomate,
American Board of Sleep Medicine

Kathleen D. Pfeffer, MD
Pediatric Sleep Specialist
Board Certification:
Pulmonary Medicine
Pediatrics
Diplomate,
American Board of Sleep Medicine

Address
9103 S. 1300 W.
Suite 103
West Jordan, UT 84088
801 432-8690
801 432-8681 FAX
UTSleep.com

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DOS: 9-20-13

Under these circumstances, checking Will's iron and ferritin level and making sure hypoferritinemia is not contributing to leg movements would be worthwhile. A trial of gabapentin, Clonidine, or Clonazepam before bed might also be helpful in calming his legs and allowing more sleep continuity.

Kathleen D. Pfeffer, M.D.
Diplomat, American Academy of Sleep Medicine
Board Certified Pediatric Pulmonologist

cc: Home
dm:

disorder which might contribute to Will's difficulties falling to sleep and maintaining sleep.

METHOD: The patient was monitored by a technician and studied with full, conventional polysomnography, including continuous measurements of EEG, EOG, oral/nasal airflow, single channel ECG, leg EMG, chest and abdominal movements by plethysmography and video and audio recording.

RESULTS: Overall this was a really hard study for Will. Lights were turned off at 10:52 p.m. He fell asleep in 19.5 minutes. Sleep period extended from 10:51 p.m. until 5:06 a.m., or 375 minutes. During this time he slept for 308 minutes, a sleep efficiency of 78.1%. He entered into stage 3+4 sleep normally. At about 12:50 a.m., when he should have been entering into REM sleep, he entered into stage 1 sleep. He was horribly restless at this time. He then transitioned back into stage 2, stage 3+4 sleep, but around 2:15 a.m., again associated with an attempt to enter into REM sleep he transitioned into light sleep and then woke. He did not achieve sustained sleep again until 3:30. At this time he had a very long REM cycle and entered into

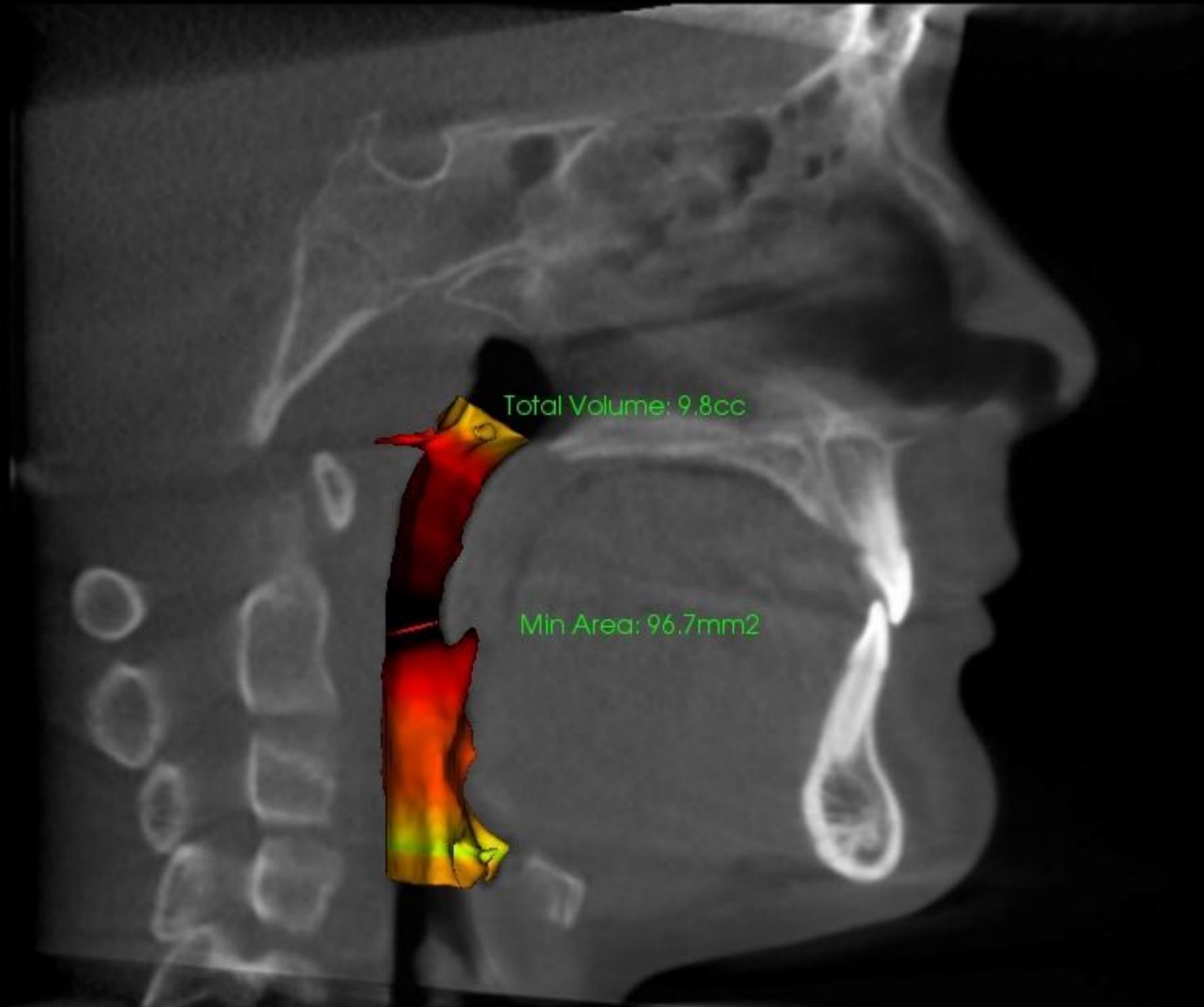
of
There were 10 central pauses also scattered throughout the night and tending to occur during transitions between deep sleep and night sleep. These averaged 11.6 seconds and lasted as long as 14.8 seconds. 3 events occurred on his back, 7 on his left side. No central apnea per se, no periodic breathing. Central apnea index (CAI) is 1.9. Overall apnea/hypopnea index (AHI) is 3.1, which is within normal limits.

4088
There were 48 RERAs or respiratory event related arousals. These are respiratory events that cannot be scored as apneas or hypopneas, yet result in arousal, and typically suggest some degree of upper airway resistance. These were also scored during periods of light stage 1 sleep, and transitions in and out of wakefulness.

Clinical information, periods of increased effort were occasionally noted in the intercostal muscles. Periods of paradoxical breathing were also occasionally seen throughout the night, although no snoring was detected. Frequent periods of tossing and turning were noted throughout the night.

IMPRESSIONS/RECOMMENDATIONS: Despite Will's feeling that his legs do not need to move, these results suggest Restless Leg Syndrome. They are not consistent with periodic limb movements, and they are not consistent with sleep

MCA = 96.7 mm²



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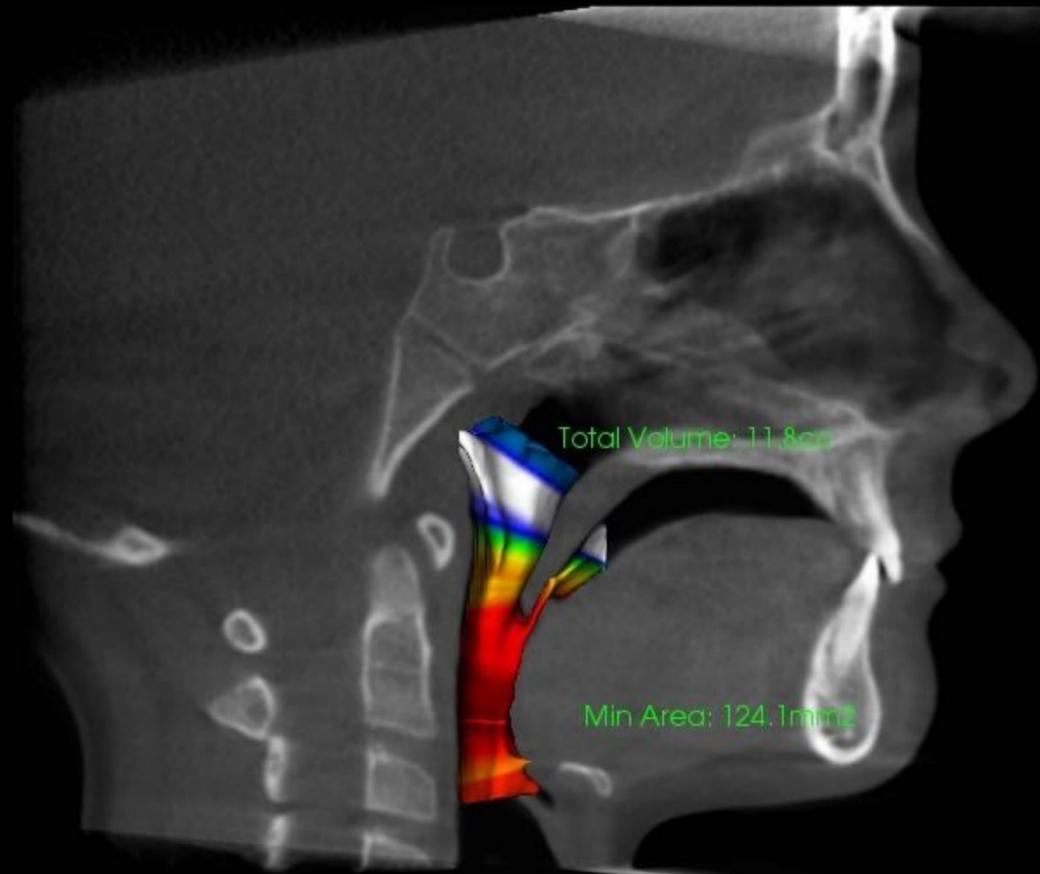
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Megan

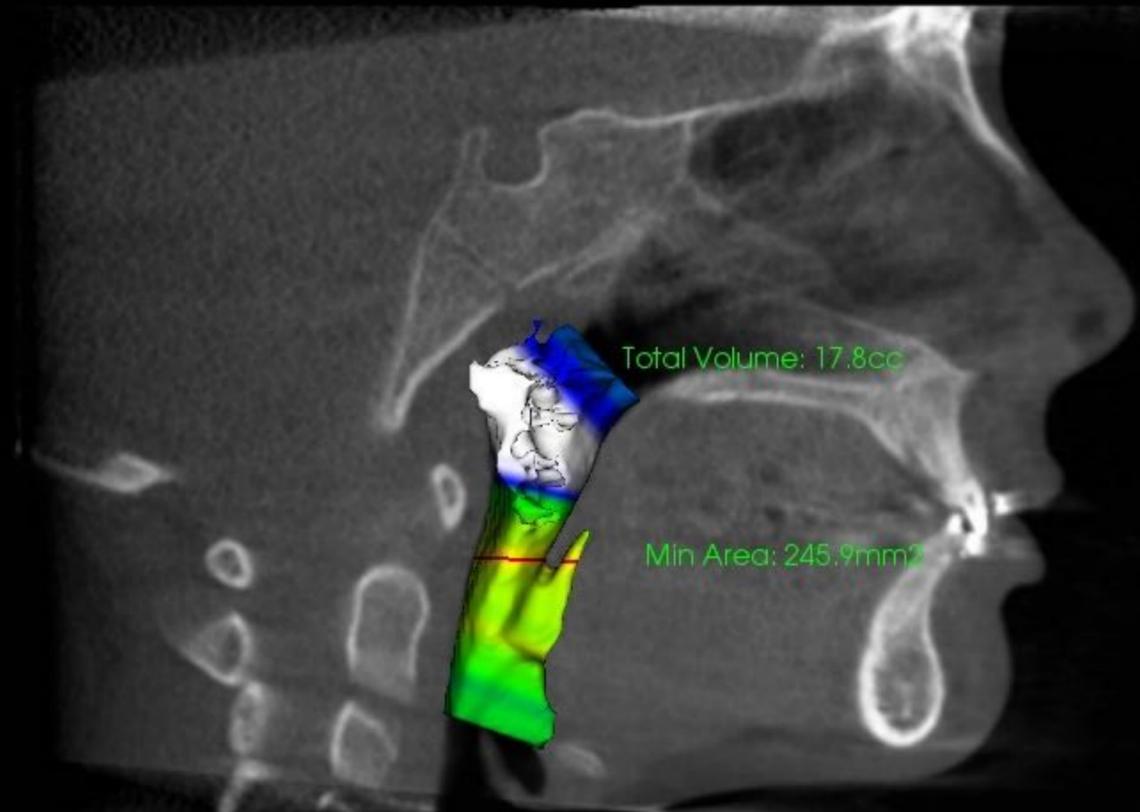
Eight months of phase 1 treatment with fixed appliances, no expander.

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MCA = 124.1 mm²



MCA = 245.9 mm²

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25TH
AADSM ANNIVERSARY MEETING
DENVER
JUNE 9 - 11, 2016

SHERATON DENVER
DOWNTOWN

The central graphic is a dark blue rectangle containing a logo. At the top, the number "25" is written in a large, stylized font, with the "2" in blue and the "5" in green. To the right of the "5" is the word "TH" in green. Below this, the text "AADSM ANNIVERSARY MEETING" is written in white, sans-serif font. A blue banner with a white border contains the word "DENVER" in large, white, serif font. Below the banner is a white mountain range silhouette with a green arc underneath it. Inside the arc, the text "JUNE 9 - 11, 2016" is written in black, with a yellow pickaxe and shovel icon below it. At the bottom of the rectangle, the text "SHERATON DENVER DOWNTOWN" is written in white, sans-serif font.

Learning Objectives

By the end of the AADSM 25th Anniversary Meeting, participants should be able to:

- 1 Acquire knowledge about the management of obstructive sleep apnea in both adults and children;
- 2 Discuss state-of-the-art knowledge of recent advances in dental sleep medicine and sleep apnea treatment;
- 3 Review the relationship between obstructive sleep apnea, cardiovascular disease and other associated co-morbidities;
- 4 Understand the evidence regarding long-term oral appliance therapy, including potential side effects and options for managing complications in patients with snoring and/or OSA; and
- 5 Apply best practices for building and developing a successful dental sleep medicine practice, including an overview of proper patient management and development of care plans; creating awareness about sleep related breathing disorders and their treatments; positioning your practice as a provider of dental sleep medicine; and proper medical insurance billing.

SCHEDULE AT A GLANCE

For full schedule details, visit www.aadsm.org/AnnualMeeting.aspx.

Thursday, June 9, 2016

8:00am-12:15pm

Educational Courses

Fee required

C01: Introduction to Dental Sleep Medicine 

Katherine Phillips, DDS, Chair | Don Farquhar, DDS | James Hogg, DDS | Kevin Postol, DDS

C02: Advanced Dental Sleep Medicine 

Alan Blanton, DDS, Chair | Marc Braem, DDS | Leslie Dort, DDS | Anne Bartolucci, PhD | Ryan Soose, MD

10:00am-4:00pm

Exhibit Hall Open

12:15pm-1:30pm 

Meet the Professors

Fee required

M01: The Role of Sleep Bruxism in Obstructive Sleep Apnea

M02: The Best Questions to Ask to Find Sleep Disorders

M03: Titration Versus Treatment Success

Ghizlane Aarab, DDS

Timothy Morgenthaler, MD

Marc Braem, DDS

1:30pm-2:15pm

Welcome Address and Awards

2:15pm-3:15pm

KEYNOTE ADDRESS



I01: Insights into the Pathogenesis and Management of OSA Utilizing Upper Airway Imaging

Richard Schwab, MD

3:15pm-3:30pm

Refreshment Break

3:30pm-4:30pm

I02: A Look Back at 25 Years of Dental Sleep Medicine

Robert Rogers, DMD

4:30pm-5:30pm

I03: Measuring Quality in the Treatment of OSA/Oral Appliances

Timothy Morgenthaler, MD

6:00pm-8:00pm

Industry Supported Events

Learning Objectives

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Friday, June 10, 2016

8:00am-9:00am	D01: Turf War: Home Sleep Apnea Testing	Gail Demko, DMD Kelly Carden, MD
	W01: Titration: Where to Start?	Ghizlane Aarab, DDS, PhD
9:00am-10:00am	S01: Year in Review	Marc Braem, DDS
	I04: PSG: What Does the Dental Sleep Clinician Need to Know?	Richard Berry, MD
10:00am-4:00pm	Exhibit Hall Open	
10:00am-10:30am	Refreshment Break and Poster Viewing	
10:30am-11:30am	I05: Sleep Apnea and Cardiovascular Disease	Atul Malhotra, MD
	W02: Telemedicine	Steve Van Hout
11:30am-12:30pm	R01: Challenging Cases	Moderator: Alan Blanton, DDS
	S02: Sleep-Disordered Breathing and Cardiometabolic Interactions in Pregnancy and in the General Population	Sushmita Pamidi, MD
12:30pm-1:00pm	ABDSM Information Session	
12:30pm-1:45pm	M04: Frequently Asked Questions about Sleep Apnea	Atul Malhotra, MD
<i>Meet the Professors</i>	M05: New Titration Protocols – How to Test Them Without Bias	Fernanda Almeida, DDS, PhD
<i>Fee required</i>	M06: The Road to Personalized Medicine for Sleep Apnea: Challenges and Opportunities	Danny Eckert, PhD
1:45pm-3:15pm	W03: PSG and HSAT: Diagnostics, Outcome Studies and Split Nights	Max Hirschkowitz, PhD
	W04: Modified Oral Appliance and Combination Therapy	Katherine Phillips, DDS James Hogg, DDS
3:15pm-3:45pm	Refreshment Break and Poster Viewing	
3:45pm-4:45pm	S03: Basic Science of Sleep, Wakefulness and Upper Airway Tone	Michael Decker, PhD
	O01: Oral Presentations	
4:45pm-5:45pm	W05: Insurance Company Audits: How to be Prepared	Mary Beth Rogers
	I06: Sleep Deprivation	David Dinges, PhD

Learning Objectives

By the end of the AADSM 25th Anniversary Meeting, participants should be able to:

- 1 Acquire knowledge about the management of obstructive sleep apnea in both adults and children;
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- 3 Review the relationship between obstructive sleep apnea, cardiovascular disease and other associated co-morbidities;
- 4 Understand the evidence regarding long-term oral appliance therapy, including potential side effects and options for managing complications in patients with snoring and/or OSA; and
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Saturday, June 11, 2016

For full schedule details, visit www.aadsm.org/AnnualMeeting.aspx.

8:00am-9:00am	S04: AADSM Accreditation: Impacting the Physician Sleep Specialist's Network	Patricia Braga, DDS Norman Blumenstock, DDS Jennifer Le, DMD
	W06: Managing OSA: CPAP vs VPAP vs AutoPAP, is there a difference?	Christopher Lettieri, MD
9:00am-10:00am	S05: Sleep and Chronic Pain	Michael Smith, MD
	S06: Midface Hypoplasia and Pediatric OSA: Causes, Correlations and Orthodontic Interventions	Soleil de Marsche Roberts, DMD
10:00am-3:00pm	Exhibit Hall Open	
10:00am-10:15am	Refreshment Break	
10:15am-11:15am	I07: Cognition, Behavior and OSA in Children	Ronald Chervin, MD, MS
	S07: Insomnia and Sleep Apnea: Collaborative Approaches to this Comorbidity	Jason Ong, PhD
11:15am-12:15pm	S08: Impact of Sleep Apnea on Neurocognition	Stuart Quan, MD
	W07: Complementary and Alternative Therapies for Insomnia Disorder	Jennifer Martin, PhD
12:15pm-1:30pm <i>Meet the Professors</i> <i>Fee required</i>	M07: Understanding the Rules and Regulations Regarding Oral Appliance Use in Pilots and Commercial Drivers	Christopher Lettieri, MD
	M08: Using Combination Therapy to Help Develop the Physician Referral Network	Patricia Braga, DDS
	M09: Sleep Medicine in 2036: Promise and Opportunities	Ronald Chervin, MD, MS
1:30pm-2:30pm	W08: Phenotyping and Oral Appliances: Towards Individualized Strategies to Optimize Treatment Success According to Underlying Mechanisms	Danny Eckert, PhD
	S09: The PTSD and Sleep Apnea Connection	Ali El Solh, MD
2:30pm-3:30pm	I08: Is Insomnia History? The Modernization of Sleep	Roger Ekirch, PhD
	W09: Objective Compliance	Fernanda Almeida, DDS, PhD
3:30pm-4:00pm	AADSM Membership Meeting	



SLEEP 2016

DENVER • JUNE 11-15



REGISTER BY APRIL 27TH TO SAVE!

30TH ANNIVERSARY MEETING OF THE ASSOCIATED
PROFESSIONAL SLEEP SOCIETIES, LLC

A JOINT MEETING OF THE



AND THE





Quality Measures for the Care of Pediatric Patients with Obstructive Sleep Apnea

Sanjeev V. Kothare, MD¹; Carol L. Rosen, MD²; Robin M. Lloyd, MD³; Shalini Paruthi, MD⁴; Sherene M. Thomas, PhD⁵; Matthew M. Troester, DO⁶; Kelly A. Carden, MD⁷

¹NYU Langone Medical Center, New York, NY; ²University Hospitals Rainbow Babies and Children's Hospital, Cleveland, OH; ³Mayo Clinic, Rochester, MN; ⁴Saint Louis University School of Medicine, St. Louis, MO; ⁵American Academy of Sleep Medicine, Darien, IL; ⁶Phoenix Children's Hospital, Phoenix, AZ; ⁷Saint Thomas Sleep Specialists, Nashville, TN

The Board of Directors of the American Academy of Sleep Medicine (AASM) commissioned a Task Force to develop quality measures as part of its strategic plan to promote high quality patient-centered care. Among many potential dimensions of quality, the AASM requested Workgroups to develop outcome and process measures to aid in evaluating the quality of care of five common sleep disorders: insomnia, obstructive sleep apnea in adults, obstructive sleep apnea in children, restless legs syndrome, and narcolepsy. This paper describes the rationale, background, general methods development, and considerations in implementation of these quality measures in obstructive sleep apnea (OSA) in children. This document describes measurement methods for five desirable process measures: assessment of symptoms and risk factors of OSA, initiation of an evidence-based action

plan, objective evaluation of high-risk children with OSA by obtaining a polysomnogram (PSG), reassessment of signs and symptoms of OSA within 12 months, and documentation of objective assessment of positive airway pressure adherence. When these five process measures are met, clinicians should be able to achieve the two defined outcomes: improve detection of childhood OSA and reduce signs and symptoms of OSA after initiation of a management plan. The AASM recommends the use of these measures as part of quality improvement programs that will enhance the ability to improve care for patients with childhood OSA.

Citation: Kothare SV, Rosen CL, Lloyd RM, Paruthi S, Thomas SM, Troester MM, Carden KA. Quality measures for the care of pediatric patients with obstructive sleep apnea. *J Clin Sleep Med* 2015;11(3):385–404.

Supporting Evidence and Rationale

There are over 70 million children between the ages of 0 and 17 years in the United States.¹⁰ Between 10% and 30% (7.4 to 22 million children) habitually snore,¹¹ and between 1% and 5% (743,000–3,715,000 children) have obstructive sleep apnea syndrome.¹ Both habitual snoring and obstructive sleep apnea syndrome have been linked to neurodevelopmental and behavioral difficulties.^{1,12,13} Data suggest that sleep disorders, including habitual snoring and obstructive sleep apnea, are under-diagnosed.¹⁴ Because these sleep disorders are treatable when recognized, it is imperative to improve their detection and ultimately reduce the associated neurodevelopmental consequences.

Normative Data

Volumetric Airway Norms

Table 1. AVERAGE AIRWAY VOLUME

Volume	Age Group (yr)													Average	SD
	6-8	9-11	12-14	15-17	18-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55	≥56		
Average	7.18	8.39	11.62	14.83	14.90	14.77	15.59	14.78	14.37	14.24	14.07	13.97	13.06	13.21	2.61
Average SD	3.40	3.45	4.79	6.00	5.35	5.50	5.91	5.80	6.03	7.96	5.42	5.50	5.42	5.42	1.15
Female															
Average	6.45	8.75	11.54	13.03	14.61	15.07	17.93	15.44	14.85	12.98	13.95	13.81	12.13	13.12	2.97
Average SD	3.13	3.39	4.30	4.45	4.98	5.90	6.14	5.75	5.74	5.17	5.26	4.79	4.98	4.92	0.92
Male															
Average	7.88	7.96	11.69	16.70	15.22	14.47	15.58	16.65	13.51	15.61	14.28	14.29	14.01	13.68	2.88
Average SD	3.54	3.51	5.19	6.84	5.79	5.11	5.43	5.79	6.47	10.03	5.74	6.73	5.75	5.84	1.62

Schendel et al. 3D Analysis of Airway Growth. J Oral Maxillofac Surg 2012.

1300 individuals

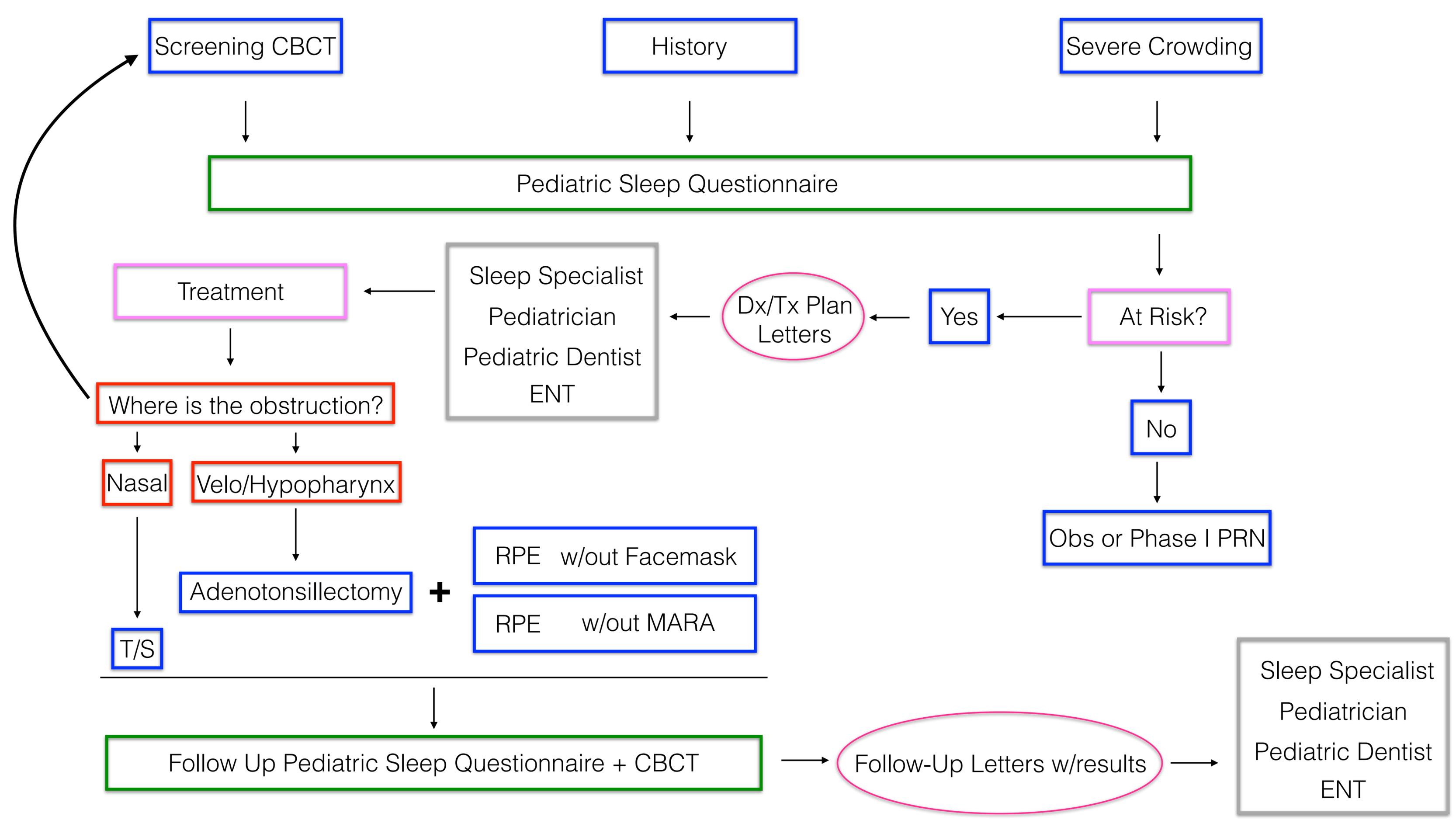
Volumetric Airway Norms

Table 4. AVERAGE SMALLEST AIRWAY AREA

Smallest Airway Area	Age Group (yr)													Average	SD
	6-8	9-11	12-14	15-17	18-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55	≥56		
Average	77.70	89.89	128.64	169.13	171.55	160.97	172.18	159.22	157.31	149.26	144.39	143.18	121.83	141.94	30.15
Average SD	48.78	47.77	66.31	86.19	113.98	80.97	81.28	81.68	84.54	120.83	75.44	81.66	82.15	80.89	20.63
Female															
Average	70.92	95.38	138.21	150.16	185.97	184.49	177.77	168.70	171.63	137.98	150.29	146.19	107.28	145.00	35.48
Average SD	44.29	49.10	70.00	72.98	140.55	94.27	84.86	80.33	86.31	75.44	74.45	79.61	65.99	78.32	23.32
Male															
Average	84.21	83.47	120.80	188.87	155.92	137.53	159.15	143.06	132.92	161.48	134.34	137.33	136.99	136.62	29.03
Average SD	52.35	45.85	62.68	94.84	73.82	56.89	71.90	82.53	76.51	155.84	77.08	86.44	94.46	79.32	27.56

Schendel et al. 3D Analysis of Airway Growth. J Oral Maxillofac Surg 2012.

1300 individuals



Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials

Gordon C S Smith, Jill P Pell

Abstract

Objectives To determine whether parachutes are effective in preventing major trauma related to gravitational challenge.

Design Systematic review of randomised controlled trials.

Data sources: Medline, Web of Science, Embase, and the Cochrane Library databases; appropriate internet sites and citation lists.

Study selection: Studies showing the effects of using a parachute during free fall.

Main outcome measure Death or major trauma, defined as an injury severity score > 15 .

Results We were unable to identify any randomised controlled trials of parachute intervention.

Conclusions As with many interventions intended to prevent ill health, the effectiveness of parachutes has not been subjected to rigorous evaluation by using randomised controlled trials. Advocates of evidence based medicine have criticised the adoption of interventions evaluated by using only observational data. We think that everyone might benefit if the most radical protagonists of evidence based medicine organised and participated in a double blind, randomised, placebo controlled, crossover trial of the parachute.

Introduction

accepted intervention was a fabric device, secured by strings to a harness worn by the participant and released (either automatically or manually) during free fall with the purpose of limiting the rate of descent. We excluded studies that had no control group.

Definition of outcomes

The major outcomes studied were death or major trauma, defined as an injury severity score greater than 15.⁶

Meta-analysis

Our statistical approach was to assess outcomes in parachute and control groups by odds ratios and quantified the precision of estimates by 95% confidence intervals. We chose the Mantel-Haenszel test to assess heterogeneity, and sensitivity and subgroup analyses and fixed effects weighted regression techniques to explore causes of heterogeneity. We selected a funnel plot to assess publication bias visually and Egger's and Begg's tests to test it quantitatively. Stata software, version 7.0, was the tool for all statistical analyses.

Results

Our search strategy did not find any randomised controlled trials of the parachute.

Discussion

Department of Obstetrics and Gynaecology, Cambridge University, Cambridge CB2 2QQ

Gordon C S Smith
professor

Department of Public Health, Greater Glasgow NHS Board, Glasgow G3 8YU

Jill P Pell
consultant

Correspondence to:
G C S Smith
gcss2@cam.ac.uk

BMJ 2003;327:1459-61

organised and participated in a double blind, randomised, placebo controlled, crossover trial of the parachute.

Introduction

The parachute is used in recreational, voluntary sector, and military settings to reduce the risk of orthopaedic, head, and soft tissue injury after gravitational challenge, typically in the context of jumping from an aircraft. The perception that parachutes are a successful intervention is based largely on anecdotal evidence. Observational data have shown that their use is associated with morbidity and mortality, due to both failure of the intervention^{1 2} and iatrogenic complications.³ In addition, “natural history” studies of free fall indicate that failure to take or deploy a parachute does not inevitably result in an adverse outcome.⁴ We therefore undertook a systematic review of randomised controlled trials of parachutes.

Methods

Literature search

We conducted the review in accordance with the QUOROM (quality of reporting of meta-analyses) guidelines.⁵ We searched for randomised controlled trials of parachute use on Medline, Web of Science, Embase, the Cochrane Library, appropriate internet sites, and citation lists. Search words employed were “parachute” and “trial.” We imposed no language restriction and included any studies that entailed jumping from a height greater than 100 metres. The

Our search strategy did not find any randomised controlled trials of the parachute.

Discussion

Evidence based pride and observational prejudice

It is a truth universally acknowledged that a medical intervention justified by observational data must be in want of verification through a randomised controlled



Parachutes reduce the risk of injury after gravitational challenge, but their effectiveness has not been proved with randomised controlled trials

trial. Observational studies have been tainted by accusations of data dredging, confounding, and bias.⁷ For example, observational studies showed lower rates of ischaemic heart disease among women using hormone replacement therapy, and these data were interpreted as advocating hormone replacement for healthy women, women with established ischaemic heart disease, and women with risk factors for ischaemic heart disease.⁸ However, randomised controlled trials showed that hormone replacement therapy actually increased the risk of ischaemic heart disease,⁹ indicating that the apparent protective effects seen in observational studies were due to bias. Cases such as this one show that medical interventions based solely on observational data should be carefully scrutinised, and the parachute is no exception.

Natural history of gravitational challenge

The effectiveness of an intervention has to be judged relative to non-intervention. Understanding the natural history of free fall is therefore imperative. If failure to use a parachute were associated with 100% mortality then any survival associated with its use might be considered evidence of effectiveness. However, an adverse outcome after free fall is by no means inevitable. Survival has been reported after gravitation challenges of more than 10 000 metres (33 000 feet).⁴ In addition, the use of parachutes is itself associated with morbidity and mortality.^{1-3 10} This is in part due to failure of the intervention. However, as with all interventions, parachutes are also associated with iatrogenic complications.³ Therefore, studies are required to calculate the balance of risks and benefits of parachute use.

What is already known about this topic

Parachutes are widely used to prevent death and major injury after gravitational challenge

Parachute use is associated with adverse effects due to failure of the intervention and iatrogenic injury

Studies of free fall do not show 100% mortality

What this study adds

No randomised controlled trials of parachute use have been undertaken

The basis for parachute use is purely observational, and its apparent efficacy could potentially be explained by a “healthy cohort” effect

Individuals who insist that all interventions need to be validated by a randomised controlled trial need to come down to earth with a bump

technology to provide effective protection against occasional adverse events.

Parachutes and the military industrial complex

However sinister doctors may be, there are powers at large that are even more evil. The parachute industry has earned billions of dollars for vast multinational corporations whose profits depend on belief in the efficacy of their product. One would hardly expect these vast commercial concerns to have the bravery to test their product in the setting of a randomised

The parachute and the healthy cohort effect

One of the major weaknesses of observational data is the possibility of bias, including selection bias and reporting bias, which can be obviated largely by using randomised controlled trials. The relevance to parachute use is that individuals jumping from aircraft without the help of a parachute are likely to have a high prevalence of pre-existing psychiatric morbidity. Individuals who use parachutes are likely to have less psychiatric morbidity and may also differ in key demographic factors, such as income and cigarette use. It follows, therefore, that the apparent protective effect of parachutes may be merely an example of the “healthy cohort” effect. Observational studies typically use multivariate analytical approaches, using maximum likelihood based modelling methods to try to adjust estimates of relative risk for these biases. Distasteful as these statistical adjustments are for the cognoscenti of evidence based medicine, no such analyses exist for assessing the presumed effects of the parachute.

The medicalisation of free fall

It is often said that doctors are interfering monsters obsessed with disease and power, who will not be satisfied until they control every aspect of our lives (*Journal of Social Science*, pick a volume). It might be argued that the pressure exerted on individuals to use parachutes is yet another example of a natural, life enhancing experience being turned into a situation of fear and dependency. The widespread use of the parachute may just be another example of doctors’ obsession with disease prevention and their misplaced belief in unproved

controlled trial. Moreover, industry sponsored trials are more likely to conclude in favour of their commercial product,¹¹ and it is unclear whether the results of such industry sponsored trials are reliable.

A call to (broken) arms

Only two options exist. The first is that we accept that, under exceptional circumstances, common sense might be applied when considering the potential risks and benefits of interventions. The second is that we continue our quest for the holy grail of exclusively evidence based interventions and preclude parachute use outside the context of a properly conducted trial. The dependency we have created in our population may make recruitment of the unenlightened masses to such a trial difficult. If so, we feel assured that those who advocate evidence based medicine and criticise use of interventions that lack an evidence base will not hesitate to demonstrate their commitment by volunteering for a double blind, randomised, placebo controlled, crossover trial.

Contributors: GCSS had the original idea. JPP tried to talk him out of it. JPP did the first literature search but GCSS lost it. GCSS drafted the manuscript but JPP deleted all the best jokes. GCSS is the guarantor, and JPP says it serves him right.

Funding: None.

Competing interests: None declared.

Ethical approval: Not required.

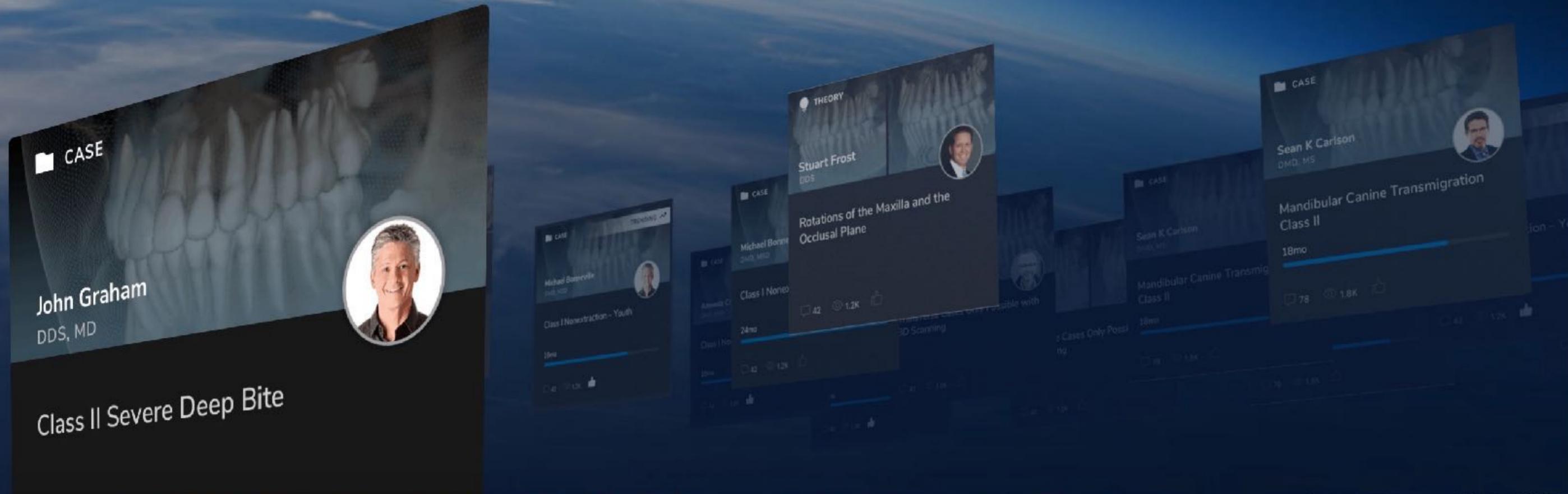
- 1 Belmont PJ Jr, Taylor KE, Mason KT, Shawen SB, Polly DW Jr, Klemme WR. Incidence, epidemiology, and occupational outcomes of thoracolumbar fractures among US Army aviators. *J Trauma* 2001;50:855-61.
- 2 Bricknell MC, Craig SC. Military parachuting injuries: a literature review. *Occup Med (Lond)* 1999;49:17-26.



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